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THE MEDICAL CLINICS of NORTH AMERICA.

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SYMPOSIUM ON PSYCHIATRY AND NEUROLOGY

FOREWORD

It is a common observation that the tremendous growth of medicine in the past few decades has been largely due to the knowledge derived from sound experimental observation. This knowledge has come to a great extent from the laboratory and has been the result of the application of scientific method to the investigation of disease. Intensive studies in the fields of chemistry, physics, mathematics and in all the biological sciences as they bear on the practice of medicine have raised the practice from the realm of art or applied science to that of a true scientific discipline. Most of diagnosis and much of treatment can truly be said now to be scientific. With the accumulation of more facts, with further progress in experimental investigations, more and more gaps in present medical knowledge will be filled, and there is reason to believe that all of medicine, preventive as well as curative, will ultimately become scientific.

The recognition of these facts, as well as their common acceptance, has led to some curious, if not erroneous, conclusions. Because the laboratory has yielded such brilliant results and given rise to such extraordinary advances, the notion is current that only that which comes from the test tube, to use a colloquial phrase, is scientific medicine. So deep-rooted has this idea become that clinical studies, if not disparaged or disdained, are looked upon with good-natured condescension. The erroneous conclusion is that clinical medicine is not scientific, while laboratory medicine is. Without detracting in the least from the scientific merits of laboratory medicine, one must recognize that clinical observation not only can be but actually is just as scientific. The assertion, as any one familiar with the history of medicine knows, can be made without fear of contradiction that not only has clinical observation served to lay the foundation for practi-

cally all of medicine, but has resulted in innumerable brilliant contributions. Accurate observation of the patient at the bedside, verification of the facts of observation, their orderly correlation, the deduction of guiding principles, and their application to practice are certainly just as scientific as laboratory experimentation. The human body is perhaps the greatest laboratory at the doctor's command—a fact too often disregarded—which beckons the observer and promises him rich scientific rewards. Actually there is no conflict between laboratory investigation and clinical observation; provided each is accurate, both are scientific.

These few prefatory remarks, valid in themselves and applicable in general, have, I believe, some relevance to the contents of the volume before us. The clinics presented herein serve the useful purpose of bringing to the reader the fruits of rich medical experience of men who combine the scientific merits of experimental research with clinical observation. They summarize the practical knowledge gained in the study and treatment of patients in office and hospital. Each writer has embodied in his article the clinical wisdom which he derived from objective experience. They cover a wide field in the ever expanding domain of psychiatry and neurology, which should reward the reader and enrich his knowledge. They will inform the general practitioner all the more as the contributions are by men who, though specialists in their own fields, are themselves practitioners of medicine. There is no condescension here by the specialist to the general practitioner.

Having read the various articles, the editor finds it difficult to single out the numerous contributions of superior merit. It would be invidious to make comparisons. All are informative, all are good, many very good. It was, therefore, a singularly difficult task to determine the order of merit which so many shared. Let the reader decide. There is both pleasure and profit in the reading. The publishers have done well to bring out the volume.

ISRAEL S. WECHSLER
Consulting Editor

HEADACHE: AN OUTLINE FOR DIAGNOSIS AND TREATMENT

E. CHARLES KUNKLE, M.D.,* AND HAROLD G. WOLFF, M.D., F.A.C.P.†

THE physician dealing with headache aims his therapy along one or more of these three channels: a frontal attack upon the immediate source of the pain (the peripheral headache mechanism), the administration of centrally acting analgesic drugs, or the elimination of the underlying illness. To be effective, such strategy demands a recognition of both the immediate and the remote causes of the headache.

In the following sections is summarized what has been learned of the nature and meaning of headache. Common and uncommon varieties of headache are classified on the basis of their peripheral mechanisms. Through such an outline the most frequently encountered types of headache (those associated with migraine, sustained muscle contraction, or febrile illnesses) come more clearly into focus when examined against a background of the many rarer headaches with which they can be compared or contrasted. Such an arrangement, moreover, helps to define clinical features which are diagnostically useful, and to indicate the basis for selective therapy.

Essential to an analysis of headache mechanisms is the knowledge that although almost any of the structures on the surface of the head are pain-sensitive, the cranium itself, the brain parenchyma, and most of the dura and pia-arachnoid are insensitive to pain. The principal intracranial sources of headache are the large dural veins and their immediate tributaries, the main dural arteries, the circle of Willis and its major arterial branches, and the nerves carrying pain-afferents, i.e., the fifth, seventh, ninth and tenth cranial nerves, and the second and third cervical nerves.

Pain arising from stimulation of structures on the outside of the head is felt largely at the site of stimulation. Painful impulses arising from intracranial structures above the tentorium cerebelli are carried by branches of the fifth cranial nerve and are referred mainly to fronto-temporal areas; from structures below the tentorium pain is carried by

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the other cranial and upper cervical nerves previously listed and is referred to the back of the head and upper neck.

In addition to these known sources, pathways and sites of pain reference, pain may sometimes extend from the site of primary noxious stimulation to other areas of the head and neck by means of central spread, probably at the segmental, i.e., brain stem, level. Hence pain may overflow into other zones in the same and sometimes in adjacent segments, as when dental pain of high intensity in a lower molar spreads into neighboring teeth or even into the upper jaw, cheek and temple. Reflex contraction of muscles of the scalp and neck, moreover, may result from noxious impulses arising elsewhere in the head, as when suboccipital muscle spasm develops in a patient afflicted with the headache of temporal migraine or the anterior headache of acute maxillary sinusitis. Such muscle contraction, when sustained, may become painful and thus provide a source of secondary headache.

The classifications which follow are imperfect mainly in their inclusive nature, for headaches as yet inadequately analyzed are listed as well as familiar types which have been extensively explored. Accordingly, when the mechanism of a headache is as yet largely surmise, *it is printed in italics*, but if the mechanism has been well defined by experimental or clinical study, it is printed in conventional type.

HEADACHES CAUSED BY DILATATION OF CRANIAL ARTERIES

Classification:

A. Intracranial Sources:

- Headache induced by vasodilator drugs (e.g., histamine, nitrites)
- Headache associated with systemic infections, usually with fever (see also B)
- Certain migraine variants (see also B)
- Headache associated with anoxemia or CO poisoning*
- Headache induced by caffeine withdrawal*
- Headache accompanying hunger*
- Headache accompanying hypoglycemia*
- Headache accompanying "hangover"*
- Headache following a convulsion*
- Headache accompanying the acute postconcussion state*
- Certain chronic postconcussion headaches*

B. Extracranial Sources:

- Migraine headache
- Headache associated with arterial hypertension
- Certain headaches associated with systemic infections, usually with fever
- Headache induced by drugs which sharply raise systemic arterial pressure (e.g., epinephrine)*
- Headache induced during exposure to negative g (centrifugal force in the seat-to-head direction)*

Diagnosis.—Headache arising from distention of cranial arteries can be manipulated in various ways to demonstrate the arterial source of the pain. Regardless of whether intracranial or extracranial arteries are

at fault, the headache is reduced in intensity or abolished by measures designed to diminish intramural pressure, as by carotid compression or by the administration of drugs which lower systemic arterial pressure. This latter method, using such agents as histamine by intravenous injection or amyl nitrite by inhalation, is of limited diagnostic value since the induced hypotension is so transient and the return of the arterial pressure to normal or hypertensive levels is accompanied by further dilatation of the relaxed walls of cranial arteries, thus then augmenting the headache.*

There are, on the other hand, certain diagnostic tests of more practical value, these may, moreover, indicate whether intracranial or extracranial arteries are the main sources of the headache. When a headache arises principally or entirely from distention of intracranial arteries, as has been shown so clearly in subjects with headache induced by injected histamine, the pain is transiently accentuated during sudden brief straining, with the concurrent rise in systemic arterial pressure. In contrast, such headache is diminished in intensity during sustained straining or bilateral jugular compression for ten to fifteen seconds (measures which raise intracranial cerebrospinal fluid pressure and thus lend increased extramural support to the dilated intracranial arteries). Intracranial pressure can be elevated even more effectively and the headache simultaneously abolished by the intrathecal injection of normal saline under controlled conditions via a needle in the lumbar sac. This particular maneuver is not feasible for routine use.

Finally, headache stemming from dilated intracranial arteries is often easily aggravated by a mild rotary head jolt to one side and back to midposition, a stress which exerts traction by the shifting brain upon the painful arteries anchoring it at the base.

When distended branches of the external carotid are the principal source of headache, as in most instances of migraine, the headache is less readily intensified by brief straining and is essentially unaffected by sustained straining jugular compression, or even vigorous head jolts. Artificial elevation of intracranial pressure by the saline method also fails to alter such headache. On the other hand, digital compression of the principal superficial artery serving the area of headache, as the temporal or occipital vessel, often favorably affects the pain. If, however, the middle meningeal artery, the major intracranial branch of the external carotid system, contributes most of the headache, temporal artery compression will be ineffective, although pressure over

* This complication can be avoided in a different and purely physical technic for lowering cranial arterial pressure i.e. by exposure of the patient to centrifugal force in the head-to-seat direction upon a human centrifuge. It has recently been shown that during direct application of forces three times the force of gravity headaches which stem from dilated cranial arteries can be completely eliminated. So vigorous and cumbersome a method of analysis, however, is limited to special experimental studies and has no general clinical application.

the ipsilateral common carotid may still be of temporary benefit. And lastly, the parenteral administration of ergotamine tartrate, a drug which tends to constrict external carotid branches, is a useful diagnostic test. When given as described below in the discussion of treatment, the agent will usually afford pronounced or complete relief within twenty to sixty minutes.

Of the listed series of underlying causes for headache arising from dilatation of cranial arteries, the separate syndromes are usually to be recognized by the antecedent history. It should be stressed that the headache associated with many febrile systemic infections may both precede and outlast the period of actual fever, and hence is only loosely termed "fever" headache. Although this headache is usually of intracranial origin, in some patients distention of extracranial arteries is the principal mechanism.

The conventional concept of migraine as a distinct clinical entity needs to be amended. Classical migraine, featured by a periodic hemicrania of moderate or high intensity with brief preheadache visual or other sensory or motor phenomena and accompanied by nausea and vomiting, is easily recognized. Intensive laboratory study has shown that the auras usually result from transient constriction of certain branches of the internal carotid arteries and that the ensuing headache itself arises mainly from dilated external carotid branches. Since, however, some branches of the internal carotid, as the superficial frontal and supraorbital arteries, reach the surface of the head, the two carotid systems may often share in the headache of migraine. Furthermore, migraine headache in some patients has clinical features which implicate intracranial arteries in part at least, and occasional instances are noted of periodic headache, either unilateral or bilateral, with responses to analysis which indicate that the headache is principally of intracranial arterial origin. The hemicrania described by Horton as "erythromelalgia of the head" may be of this group.

The immediate mechanism which induces local distention of arterial walls in migraine and its variants, particularly when the pain is unilateral, remains obscure. In many patients, it is true, certain of the cranial arteries seem particularly susceptible to vasodilatation, for a headache resembling in location and quality one of their "spontaneous" migraine headaches may be precipitated by the injection of histamine, or may occur in association with fever, hunger, omission of a usual breakfast coffee, or a "hangover."

The usual migraine attack, however, is related to less immediate inciting factors. The whole series of phenomena is a manifestation of a diffuse disturbance in body function, including the cranial vascular tree, occurring during or following stress. The individual with migraine commonly possesses traits of character which render him vulnerable to the pressures of his daily existence. Seeking to gain security by holding to inappropriate perfectionism and inflexibility, he maintains this

pattern even though it leads him recurrently to frustration, resentment and chronic dissatisfaction. The migraine attack in some occurs during stressful experiences and in others is more often noted with change in pace or "let-down" after a period of tension.

In patients with headache associated with arterial hypertension the headache is in many ways like that of migraine, for the pain is dependent more upon the state of relaxation of the cranial arteries than upon the degree of hypertension. And as in migraine the occurrence of the headache is commonly related to emotional stresses and adverse life situations.

Disputed Concepts—In the past decade the considerable attention which has been focused upon the headache experimentally induced by histamine has aroused interest in the role histamine may play in various clinical vascular headaches. This pertinent question remains unanswered, for convincing evidence that histamine is a causative factor in any of these states has not yet been adduced. "Desensitization" to histamine has been attempted by repeated intravenous or hypodermic administration of the drug but the value of this procedure in the treatment of headache has not been conspicuous. It can fairly be said that the benefits derived are no better than those noted in response to nonspecific supportive measures supplied by the earnest and sympathetic physician.

Overemphasis has also been given to the matter of allergy and headache, particularly in individuals with migraine. It has been claimed that migraine is frequently an allergic reaction, usually to certain food substances, yet no well-controlled clinical study has yet been reported to support this contention or to prove that improvement on prescribed diets is specifically due to elimination of offending allergens. It is conceivable that food sensitivity may occasionally induce a headache, but in every such instance the assumption should be closely questioned until proven valid.

Treatment.—Among the headaches arising from distention of cranial arteries, only that of typical migraine can be effectively treated by a direct attack upon the mechanism i.e. by "end-organ" therapy. This specific approach is by means of ergotamine tartrate in 0.25 to 0.5 mg amounts given intramuscularly early in the course of the headache. With rare exceptions, no more than 0.5 mg should be administered intramuscularly in any one week. When given sublingually in 3.0 mg amounts, repeated twice more at half hour intervals, the drug is much less effective. Ergotamine is of little or no benefit to the minority of patients with atypical migraine in whom dilatation of intracranial arteries is an important component in the pain. Its use is contraindicated in pregnancy, hepatic disease, sepsis, coronary insufficiency or obliterative peripheral vascular disease. Although it may reduce the intensity of the headache associated with hypertension, its use for this purpose is inadvisable.

Except for a somewhat smaller tendency to produce unwelcome side reactions, a newer product, dihydroergotamine, has no definite advantages. Evidence that the ergot alkaloids may increase intracranial blood flow in man suggests the hypothesis that such an effect, perhaps favorably altering the function of vasomotor centers in the hypothalamus or brain stem, indirectly contributes to the value of these drugs in the treatment of migraine headache. A similar reaction may conceivably also explain the benefits reported from the use of intravenous nicotinic acid or 100 per cent oxygen by inhalation in patients with headache, but no experimental support for this conjecture is yet available. In some patients with migraine headaches, experience supports the value of a cup or two of strong coffee taken soon after the symptom begins; the benefit derived can be attributed to the mild vasoconstriction of extracranial arteries induced by caffeine when taken by mouth, although nonspecific effects may be fully as significant.

The use of analgesics in the therapy of headache has a more venerable tradition. Regardless of its source, a mild headache will usually respond favorably to 0.3 to 0.6 gm. of acetylsalicylic acid by mouth, repeated as often as every two hours if necessary. When the headache is severe, it may require codeine phosphate in 60 mg. amounts. Morphine need rarely be used and is contraindicated for patients with headaches which are likely to recur frequently.

Therapy directed at the underlying causes of cranial arterial headache is of dramatic and immediate value only in those instances of systemic infections, such as malaria or septicemia, for which specific remedies are available. Because migraine headache so frequently develops in a setting of tension and fatigue, the use of ergotamine and analgesics should be reinforced by a period of rest in a darkened room, preferably preceded by a lukewarm tub bath for twenty minutes. Even when, in response to ergotamine, the headache is quickly abolished, resumption of full activity should be deferred for at least two hours.

Of greater importance in dealing with the migrainous individual is the prevention of attacks by modification of stressful factors both in the environment and in the emotional life of the patient. External threats cannot always be evaded, but through planned interviews the patient may be helped to deal constructively with them. To this end he must identify the origins of his perfectionism and inflexibility and must understand the costly effects of such attitudes. These long-term aims need not involve the assistance of a psychoanalyst. They do, however, require a physician willing to appraise with care the essential nature of his patient and to exploit to the full the tools of brief psychotherapy. The program is well begun when the patient is brought to recognize that the sources of his headaches are neither obscure nor ominous and that the outcome of treatment is largely in his own hands.

HEADACHES CAUSED BY TRACTION UPON INTRACRANIAL STRUCTURES (CHIEFLY VASCULAR)

Classification:

- A. Headache induced by vigorous head movement ("jolt" headache)
- B. Headache associated with expanding intracranial masses
- C. Headache induced during prolonged exposure to increased positive g (centrifugal force in the head-to-seat direction)

Diagnosis.—In a foregoing section reference was made to the ease with which headache due to dilatation of intracranial branches of the internal carotid arteries can be briefly accentuated by sudden head movement. By such a jolt, further traction is exerted upon the distended arteries at the base of the brain, thus augmenting the deformity which is the source of the headache. The jolt maneuver, if vigorously performed, can itself elicit a transient anterior headache in most normal subjects free of headache at the time of test and represents the simplest example of experimental headache due to traction.

In subjects with expanding intracranial masses, whether tumor or subdural hematoma, the headache so often present is usually attributable to sustained displacement of and traction upon any or several of the pain-sensitive structures which help to anchor the brain to the cranium. The headache is rarely intense, is usually aggravated by coughing or straining (measures which distend intracranial vessels) and is often readily made worse by even mild head movement. The intensity of the headache is not diminished by compression of surface arteries of the head, nor by the administration of vasoconstrictor drugs.

There is no clinical feature of such headache which reliably indicates the presence of a space-occupying intracranial lesion. Other symptoms and signs are essential to the diagnosis. However, in a patient presumed to harbor a brain tumor: (1) if the headache was initially or entirely over the back of the head, the lesion is probably infratentorial; (2) if the headache is unilateral and papilledema is absent, the headache indicates the side on which the tumor lies; and (3) if the head pain transiently induced by head jolting is always felt on one side regardless of the direction of jolt, this is probably the side of the lesion.

Treatment.—The therapy of headache due to traction upon intracranial structures depends primarily upon the management of the underlying disease, which is usually a task for the surgeon. Morphine should be withheld, but codeine may be administered when necessary. Transient decompression results from the intravenous administration of 50 per cent glucose or sucrose, or the administration of 25 per cent magnesium sulfate slowly by proctoclysis. If the mass is not surgically removable, decompression through a cranial and dural opening may be necessary.

HEADACHES CAUSED BY TRACTION PLUS DILATATION OF INTRACRANIAL VESSELS

Classification:

- A. Headache induced by free drainage of cerebrospinal fluid ("drainage" headache)
- B. Headache following lumbar puncture ("leakage" headache; see also A under Headaches Caused by Inflammation of Pain-sensitive Structures)
- C. Headache following pneumo-encephalography (see also A under Headaches Caused by Inflammation of Pain-sensitive Structures)

Diagnosis.—Headaches in this group follow the loss of 10 per cent or more of the total cerebrospinal fluid volume during or after lumbar puncture. The headache is frontotemporal and often also suboccipital. It is characteristically present when the patient is erect and is greatly improved or abolished when he is horizontal. When he is erect, the intensity of the headache is often reduced by full flexion or full extension of the head, and is increased by bilateral jugular compression or mild head jolting. Although artificial elevation of intracranial pressure by the intrathecal injection of saline will temporarily eliminate the headache, this technic cannot be justified for routine diagnostic use. Experimental studies have indicated that the headache results from traction by the sagging brain upon dilated vascular supports, principally veins. Infrequently, postpuncture headache is associated with signs of a sterile meningitis and then, as also in patients with reactions to pneumo-encephalography, headache develops and persists even while the patient remains flat in bed and is often accompanied by some degree of nuchal rigidity and low grade fever.

Treatment.—Analgesic drugs are usually of limited value in patients with headache following loss of cerebrospinal fluid. The patient should be firmly reassured that the reaction is self-limited, although occasionally it may persist for a week or more. There is no consistently reliable method of interrupting the pain mechanism, for the parenteral administration of hypotonic solutions cannot restore the cerebrospinal fluid volume to normal if the dural-arachnoid puncture hole remains open and leakage persists. If the headache is other than mild, rest flat in bed for twenty-four hours, or longer if necessary, will usually shorten the duration of the reaction.

The occurrence of postpuncture headache can be made less likely but not predictably prevented by measures which minimize leakage of fluid after the tap. As small a needle should be used as is consistent with the purposes of the puncture and multiple perforations of the meninges should be avoided. The patient should remain immobile during the procedure, for movements of head, neck or spine have been shown to produce shifts in the position of the dura and may enlarge the needle hole and thus delay healing.

It is uncertain whether the occurrence of a postpuncture reaction

is minimized by the traditional policy of maintaining the patient on bed rest for twenty-four hours after the tap. Early ambulation after the procedure is currently advocated on the basis of reports that headache is far less commonly noted under such management (Adler; McCarty and Raney). The best policy in this matter is still debatable.

HEADACHES CAUSED BY INFLAMMATION OF PAIN-SENSITIVE STRUCTURES

Classification:

A. Intracranial Sources:

- Headache associated with meningitis
- Headache associated with subarachnoid hemorrhage
- Headache following pneumo-encephalography
- Headache following lumbar puncture (rarely)
- Headache associated with some instances of cranial arteritis*

B. Extracranial Sources:

- Headache associated with cranial arteritis (notably temporal arteritis)
- Infectious or traumatic lesions of scalp or cranial periosteum

Diagnosis.—In the disease states of this group, headache is usually a symptom of secondary importance. Especially in patients with meningitis or subarachnoid hemorrhage, the nature of the illness is indicated by a familiar series of dramatic symptoms and signs and by examination of the cerebrospinal fluid. The headache is commonly intense and is made worse by coughing or straining. When the inflammatory lesion is intracranial, the headache may be readily accentuated by even very mild head jolts.

In patients with headache associated with arteritis of the temporal or other superficial vessels, or with local lesions of the scalp or cranial periosteum, the source of the pain is manifest to inspection and palpation.

Treatment.—The high intensity of headaches in this group often requires the repeated use of codeine. When the underlying cause is bacterial meningitis, effective relief follows control of the disease by appropriate chemical or antibiotic agents. The headache of subarachnoid hemorrhage usually abates spontaneously within a few days after the bleeding ceases. The pain of superficial arteritis can sometimes be greatly diminished by excision of a segment of the involved artery.

HEADACHES CAUSED BY CONTRACTION OF SKELETAL MUSCLES OF HEAD AND NECK

Classification:

- A. Headache associated with emotional tension
- B. Headache secondary to noxious stimuli elsewhere in the head
- C. Headache associated with local muscle injury

Diagnosis.—Sustained contraction of skeletal muscles of the head and neck is an exceedingly common cause for headache. It is often

precipitated by anxiety and may develop with striking rapidity when the subject is faced with a critical stress, especially if feelings of resentment or frustration are dominant. In such settings the headache is generally associated with feelings of general bodily tension. Painful muscle contraction may also be induced secondary to pain elsewhere in the head and may outlast the primary pain. It is frequently observed as a component of migraine or chronic posttraumatic headache, and is particularly common in tense individuals with painful eye, dental, nasal or paranasal disease. And finally, muscle-contraction headache may result from local infection or injury of the muscle groups.

The headache of contracted muscles is accompanied by feelings of tightness or pressure in the involved area. It is most prominently noted when the sources are the suboccipital and upper nuchal muscles. Tenderness over these zones and throughout the trapezius is often present. Frontal headache, produced by contraction of the frontalis muscle, is less common and is of low intensity. Headache of these types may be slightly accentuated by movements which stretch the taut muscles, and is reduced in intensity by physiotherapeutic measures which promote muscle relaxation. The pain is not significantly altered by any of the various tests for vascular headache described in earlier sections.

Treatment.—In addition to the usual analgesics, treatment may include bed rest, heat, massage and, when necessary, head traction. When the muscle contraction is related to life stress and emotional tension, the psychotherapeutic approach outlined in the section on migraine must be used. Reassurance, full discussion and reeducation are the aims of treatment.

HEADACHES CAUSED BY SPREAD OF PAIN FROM DISEASE ELSEWHERE IN HEAD

Classification:

- A. "Ice-cream" headache
- B. Headache associated with disease of the eye, nose and paranasal structures, ears, or teeth

Diagnosis.—Spread of pain from a site of noxious stimulation in the head is seen in perhaps its purest form in the curious but clinically unimportant "ice-cream" headache. This is the familiar brief midfrontal ache which may follow contact of ice-cold foods against the roof of the mouth. An analogous mechanism explains the anterior component of headaches sometimes noted in association with disease of the eyes, nose, paranasal sinuses, teeth or ears. Contrary to the almost universal lay impression, however, only a small proportion of headaches are attributable to these head structures. Because of secondary nuchal muscle contraction, as described in the section on Headaches Caused by Contraction of Skeletal Muscles of Head and

Neck, a posterior component is frequently added to the anterior headache.

The types of ocular defect which may induce headache are chiefly glaucoma, iritis, hypermetropia, astigmatism and marked degrees of extraocular imbalance. The headache is mainly orbital and frontal. It usually begins in the latter half of the day.

Headache associated with disease of the nasal and paranasal structures is felt primarily over the malar, nasal or frontal areas. Because of engorgement of the turbinates the headache is commonly intensified by procedures which increase venous pressure locally, as in coughing or straining, or which displace the turbinates, as in sudden head movement. The headache usually begins and reaches its peak soon after arising in the morning and improves late in the day. Two axioms have practical value in diagnosis: (1) if headache is unassociated with inflammation and turgescence of the nasal mucosa, it is probably not caused by nasal or sinus disease; and (2) if anterior headache is unrelieved by the intranasal application of vasoconstrictor agents and cocaineization of the ostia to the sinuses, it is probably not caused by nasal or sinus disease.

When headache is produced by pain overflowing from a diseased tooth or ear, the primary pain is so prominent that the site of the responsible lesion can scarcely be overlooked.

Treatment.—Headaches resulting from disease of any of these structures are managed by the use of analgesics and by correction of the primary defects.

"PSYCHOGENIC" HEADACHE

Diagnosis.—In a small percentage of patients with headache the symptom cannot be interpreted in terms of end-organ mechanisms and has features which indirectly indicate that it is elaborated at supra-segmental levels. Such headache is often loosely termed "psychogenic." It is here so designated in a highly limited sense, to contrast it with the host of other headaches for which some peripheral structural change (either reversible or irreversible) is responsible. All of the headaches thus far discussed, including those stemming from personality disturbances, are literally "physiogenic." The "psychogenic" remnant is segregated only because the pathophysiology of such headache, or indeed of other symptoms conventionally termed hysterical, is as yet incompletely defined.

"Psychogenic" headache may be protracted and disabling because of the associated reaction pattern of the host, but seems usually to be of only moderate intensity. In some patients it is actually described as a sense of pressure rather than a true pain. It is often bizarre in quality and location, as when it is termed burning or pricking, and centers over the vertex or shifts perversely from area to area. The responses of the headache to the diagnostic tests previously outlined may be

HYPNOTIC PSYCHOTHERAPY

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SINCE the most primitive times hypnosis has been employed almost universally in the practice of religious and medical rites to intensify belief in mysticism, magic and medicine. The impressive bewildering character of hypnotic manifestations and the profoundly inexplicable, seemingly miraculous, psychological effects upon human behavior achieved by the use of hypnosis, have served to bring about two general contradictory attitudes toward it. The first of these is the unscientific attitude of superstitious awe, fear, disbelief and actual hostility, all of which have delayed and obstructed the growth of scientific knowledge of hypnosis.

The second attitude is one of scientific acceptance of hypnosis as a legitimate and valid psychological phenomenon, of profound importance and significance in the investigation and understanding of human behavior, and of the experiential life of the individual. This attitude had its first beginnings with the work of Anton Mesmer in 1775, who tempered his scientific approach to an understanding of hypnosis by mystical theories. Nevertheless, Mesmer did succeed in demonstrating the usefulness and effectiveness of hypnosis in the treatment of certain types of patients otherwise unresponsive to medical care. Thus he laid the foundation for the therapeutic use of hypnosis and for the recognition of psychotherapy as a valid psychological medical procedure.

Since then there has been a long succession of clinically trained men who demonstrated the usefulness of hypnosis as a therapeutic medical procedure and as a means of examining, understanding and reeducating human behavior. Among these was James Braid, a Scotch physician who, in 1841, first discredited the superstitious mystical ideas about the nature of hypnosis, or "mesmerism," as it was then called. Braid recognized the phenomenon as a normal psychological manifestation, coined the terms of "hypnosis" and "hypnotism," and devised a great variety of scientific experimental studies to determine its medical and psychological values.

Following Braid, many outstanding scientists, including both clinicians and later psychologists, accepted his findings and contributed increasingly to the scientific development of hypnosis despite the

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hampering heritage of traditional misconceptions, fears and hostilities that have surrounded it and still do among the uninformed.

As yet, a scientific knowledge of hypnosis is still in its infancy. Theories of its nature are too general and too inadequate. Methods of application constitute a problem warranting extensive investigation. A general appreciation of the need to integrate hypnotic studies with current knowledge is only slowly developing. The types of disorders for which it is best suited are still undetermined. New variations in technics need to be developed.

As for the utilization of hypnosis in psychotherapy, this, too, is still in its infancy. Traditions and traditional ways of thinking, the rigid self-sufficiency of various schools of psychotherapy, and the human tendency to fear the new and untried have hampered studies in this field. Only during the past twenty-five years has there been an increasing number of studies demonstrating hypnosis to be of outstanding value in investigating the nature and structure of the personality, in understanding normal and abnormal behavior, in studying interpersonal and intrapersonal relationships and psychosomatic interrelationships. Also, there have been extensive developments in the utilization of hypnosis as an effective instrument for psychotherapy. During the second World War there was a tremendous increase in the recognition and utilization of hypnosis as a valuable form of psychotherapy.

Any discussion of hypnotic psychotherapy or hypnotherapy requires an explication of certain general considerations derived directly from clinical observation. In the following pages an effort will be made to indicate some of the misconceptions, inadequate understandings, oversights, and failures of differentiation which hamper or militate against the acceptance and usefulness of hypnotherapy. Also, material will be given to illustrate various technics, and explanations of their use will be given.

DIFFERENTIATION BETWEEN TRANCE INDUCTION AND TRANCE STATE

One of the first considerations in undertaking hypnotic psychotherapy centers around the differentiation of the patient's experience of having a trance induced from the experience of being in a trance state. As an analogy, the train trip to the city is one order of experience; being in the city is another. To continue, the process of inducing a trance should be regarded as a method of teaching the patient a new manner of learning something, and thereby enabling him to discover unrealized capacities to learn, and to act in new ways which may be applied to other and different things. The importance of trance induction as an educational procedure in acquainting the patient with his latent abilities has been greatly disregarded.

Both the therapist and the patient need to make this differentiation, the former in order to guide the patient's behavior more effectively,

the latter in order to learn to distinguish between his conscious and unconscious behavior patterns. During trance induction, the patient's behavior is comprised of both conscious and unconscious patterns, while the behavior of the trance state should be primarily of unconscious origin.

The failure of such distinction or differentiation between the induction and the trance often results in the patient's attempting to perform the work of the trance state in the same fashion as he learned to develop a trance. That is, without proper differentiation, the patient will utilize both conscious and unconscious behavior in the trance instead of relying primarily upon unconscious patterns of behavior. This leads to inadequate faulty task performance.

Although patients can, and frequently do, make this distinction spontaneously, the responsibility, though often overlooked, rests properly with the therapist. To insure such differentiation, the trance induction should be emphasized as a preparation of the patient for another type of experience in which new learnings will be utilized for other purposes and in a different way. This education of the patient can be achieved best, as experience has shown, by teaching him how to become a good hypnotic subject, familiar with all types of hypnotic phenomena. This should be done before any attempt is made at therapy. Such training, while it postpones the initiation of direct therapy, actually hastens the progress of therapy since it gives the patient wider opportunities for self-expression. For example, the patient who can develop hypnotic hallucinations, both visual and auditory, manifest regressive behavior, do automatic writing, act upon post-hypnotic suggestions, and dream upon command is in an advantageous position for the reception of therapy.

As for the trance state itself, this should be regarded as a special, unique, but wholly normal psychological state. It resembles sleep only superficially, and it is characterized by various physiological concomitants, and by a functioning of the personality at a level of awareness other than the ordinary or usual state of awareness. For convenience in conceptualization, this special state, or level of awareness, has been termed "unconscious" or "subconscious." The role in hypnotic psychotherapy of this special state of awareness is that of permitting and enabling the patient to react, uninfluenced by his conscious mind, to his past experiential life and to a new order of experience which is about to occur as he participates in the therapeutic procedure. This participation in therapy by the patient constitutes the primary requisite for effective results.

ROLE OF SUGGESTION IN HYPNOSIS

The next consideration concerns the general role of suggestion in hypnosis. Too often, the unwarranted and unsound assumption is made that, since a trance state is induced and maintained by suggestion,

and since hypnotic manifestations can be elicited by suggestion, whatever develops from hypnosis must necessarily be completely a result of suggestion and primarily an expression of it.

Contrary to such misconceptions, the hypnotized person remains the same person. His behavior only is altered by the trance state, but even so, that altered behavior derives from the life experience of the patient and not from the therapist. At the most, the therapist can influence only the manner of self-expression. The induction and maintenance of a trance serve to provide a special psychological state in which the patient can reassociate and reorganize his inner psychological complexities and utilize his own capacities in a manner in accord with his own experiential life. Hypnosis does not change the person nor does it alter his past experiential life. It serves to permit him to learn more about himself and to express himself more adequately.

Direct suggestion is based primarily, if unwittingly, upon the assumption that whatever develops in hypnosis derives from the suggestions given. It implies that the therapist has the miraculous power of effecting therapeutic changes in the patient, and disregards the fact that therapy results from an inner resynthesis of the patient's behavior achieved by the patient himself. It is true that direct suggestion can effect an alteration in the patient's behavior and result in a symptomatic cure, at least temporarily. However, such a "cure" is simply a response to the suggestion and does not entail that reassociation and reorganization of ideas, understandings and memories so essential for an actual cure. It is this experience of reassociating and reorganizing his own experiential life that eventuates in a cure, not the manifestation of responsive behavior which can, at best, satisfy only the observer.

For example, anesthesia of the hand may be suggested directly and a seemingly adequate response may be made. However, if the patient has not spontaneously interpreted the command to include a realization of the need for inner reorganization, that anesthesia will fail to meet clinical tests and will be a pseudo-anesthesia.

An effective anesthesia is better induced, for example, by initiating a train of mental activity within the patient himself by suggesting that he recall the feeling of numbness experienced after a local anesthetic, or after a leg or arm went to sleep, and then suggesting that he can now experience a similar feeling in his hand. By such an indirect suggestion the patient is enabled to go through those difficult inner processes of disorganizing, reorganizing, reassociating and projecting of inner real experience to meet the requirements of the suggestion and thus, the induced anesthesia becomes a part of his experiential life, instead of a simple, superficial response.

The same principles hold true in psychotherapy. The chronic alcoholic can be induced by direct suggestion to correct his habits temporarily, but not until he goes through the inner process of reassociating and reorganizing his experiential life can effective results occur.

In other words, hypnotic psychotherapy is a learning process for the patient, a procedure of reeducation. Effective results in hypnotic psychotherapy, or hypnotherapy, derive only from the patient's activities. The therapist merely stimulates the patient into activity, often not knowing what that activity may be, and then he guides the patient and exercises clinical judgment in determining the amount of work to be done to achieve the desired results. How to guide and to judge constitute the therapist's problem while the patient's task is that of learning through his own efforts to understand his experiential life in a new way. Such reeducation is, of course, necessarily in terms of the patient's life experiences, his understandings, memories, attitudes and ideas, and it cannot be in terms of the therapist's ideas and opinions. For example, in training a gravid patient to develop anesthesia for eventual delivery, use was made of the suggestions outlined above as suitable. The attempt failed completely even though she had previously experienced local dental anesthesia and also her legs "going to sleep." Accordingly, the suggestion was offered that she might develop a generalized anesthesia in terms of her own experiences when her body was without sensory meaning to her. This suggestion was intentionally vague since the patient, knowing the purpose of the hypnosis, was enabled by the vagueness of the suggestion to make her own selection of those items of personal experience that would best enable her to act upon the suggestion.

She responded by reviewing mentally the absence of any memories of physical stimuli during physiological sleep, and by reviewing her dreams of walking effortlessly and without sensation through closed doors and walls and floating pleasantly through the air as a disembodied spirit looking happily down upon her sleeping, unfeeling body. By means of this review, she was able to initiate a process of reorganization of her experiential life. As a result, she was able to develop a remarkably effective anesthesia, which met fully the needs of the subsequent delivery. Not until sometime later did the therapist learn by what train of thought she had initiated the neuro-psycho-physiological processes by which she achieved anesthesia.

SEPARATENESS OF CONSCIOUS AND SUBCONSCIOUS LEVELS OF AWARENESS

Another common oversight in hypnotic psychotherapy lies in the lack of appreciation of the separateness or the possible mutual exclusiveness of the conscious and the unconscious (or subconscious) levels of awareness. Yet, all of us have had the experience of having a word or a name "on the tip of the tongue" but being unable to remember it so that it remained unavailable and inaccessible in the immediate situation. Nevertheless, full knowledge actually existed within the unconscious, but unavailably so to the conscious mind.

In hypnotic psychotherapy, too often, suitable therapy may be given

to the unconscious but with the failure by the therapist to appreciate the tremendous need of either enabling the patient to integrate the unconscious with the conscious, or, of making the new understandings of the unconscious fully accessible, upon need, to the conscious mind. Comparable to this failure would be an appendectomy with failure to close the incision. It is in this regard that many arm-chair critics naively denounce hypnotic psychotherapy as without value since "it deals only with the unconscious." Additionally, there is even more oversight of the fact, repeatedly demonstrated by clinical experience, that in some aspects of the patient's problem direct reintegration under the guidance of the therapist is desirable; in other aspects, the unconscious should merely be made available to the conscious mind, thereby permitting a spontaneous reintegration free from any immediate influence by the therapist. Properly, hypnotherapy should be oriented equally about the conscious and unconscious, since the integration of the total personality is the desired goal in psychotherapy.

However, the above does not necessarily mean that integration must constantly keep step with the progress of the therapy. One of the greatest advantages of hypnotherapy lies in the opportunity to work independently with the unconscious without being hampered by the reluctance, or sometimes actual inability, of the conscious mind to accept therapeutic gains. For example, a patient had full unconscious insight into her periodic nightmares of an incestuous character from which she suffered, but, as she spontaneously declared in the trance, "I now understand those horrible dreams, but I couldn't possibly tolerate such an understanding consciously." By this utterance, the patient demonstrated the protectiveness of the unconscious for the conscious. Utilization of this protectiveness as a motivating force enabled the patient subsequently to accept consciously her unconscious insights.

Experimental investigation has repeatedly demonstrated that good unconscious understandings allowed to become conscious before a conscious readiness exists will result in conscious resistance, rejection, repression and even the loss, through repression, of unconscious gains. By working separately with the unconscious there is then the opportunity to temper and to control the patient's rate of progress and thus to effect a reintegration in the manner acceptable to the conscious mind.

ILLUSTRATIVE CASE HISTORY

A 28 year old married man sought therapy because he believed implicitly that he did not love his wife and that he had married her only because she resembled superficially his mother to whom he was strongly attached. In the trance state he affirmed this belief. During hypnotherapy, he learned, in the trance state, that his marital problem had arisen from an intense mother-hatred disguised as oversolicitude and that his wife's superficial resemblance to the mother rendered her an excellent target for his manifold aggressions. Any attempt to make his unconscious under-

standings conscious confronted him with consciously unendurable tasks of major revisions in all of his interpersonal relationships and a recognition of his mother-hatred which, to him, seemed to be both intolerable and impossible.

In psychotherapy, other than hypnotic, the handling by the patient of such a problem as this would meet with many conscious resistances, repressions, rationalizations and efforts to reject any insight. The hypnotic therapeutic procedures employed to correct this problem will be given in some detail below. No attempt will be made to analyze the underlying dynamics of the patient's problem since the purpose of this paper is to explicate methods of procedure, new technics, the utilization of mental mechanisms, and the methods of guiding and controlling the patient's progress so that unconscious insight becomes consciously acceptable.

Early in the course of this patient's treatment it had been learned that he did not consciously dare to look closely at his mother, that he did not know the color of his mother's eyes, or the fact that she wore dentures, and that a description of his mother was limited to "she is so gentle and graceful in her movements, and her voice is so soft and gentle, and she has such a sweet, kind, gracious expression on her face that a miserable neurotic failure like me does not deserve all the things she has done for me."

When, during hypnotherapy, he had reached a stage at which his unconscious understandings and insights seemed to be reasonably sufficient to permit the laying of a foundation for the development of conscious understandings, he was placed in a profound somnambulistic trance. He was then induced to develop a profound amnesia for all aspects of his problem, and a complete amnesia for everything about his mother and his wife, except the realization that he must have had a mother. This amnesia included also his newly acquired unconscious understandings.

There are special reasons for the induction of such a profound amnesia or repression. One is that obedience to such a suggestion constitutes a relinquishing of control to the therapist of the patient's repression tendencies. Also, it implies to the patient that if the therapist can repress he also can restore. In undertaking hypnotherapy, it is important in the early stages to have the patient develop an amnesia for some innocuous memory, then to restore that memory along with some other unimportant but forgotten memory. Thus, an experiential background is laid for the future recovery of vital repressed material.

The other reason is that such an amnesia or induced repression clears the slate for a reassociation and reorganization of ideas, attitudes, feelings, memories and experiences. In other words, the amnesia enables the patient to be confronted with material belonging to his own experiential life but which, because of the induced repression, is not recognized by him as such. Then it becomes possible for that patient to reach a critical objective understanding of unrecognized material

from his own life experience, to reorganize and reassociate it in accord with its reality significances and his own personality needs. Even though the material has been repressed from both the unconscious and the conscious, his personality needs still exist and any effort to deal with the material presented will be in relationship to his personality needs. As an analogy, the child on a calcium deficient diet knows nothing about calcium deficiency nor calcium content, but, nevertheless, shows a marked preference for calcium rich food.

After the induction of the amnesia, the next step was a seemingly casual brief discussion of the meaning of feminine names. Then it was suggested that he see, sitting in the chair on the other side of the room, a strange woman who would converse with him and about whom he would know nothing except for a feeling of firm conviction that her first name was Nelly. Previous hypnotic training at the beginning of hypnotherapy had prepared him for this type of experience.

The patient's response to that particular name, as intended, was that of an hallucination of his mother whom he could not, because of the amnesia, recognize as such. He was induced to carry on an extensive conversation with this hallucinatory figure, making many inquiries along lines pertinent to his own problem. He described her adequately and objectively. He was asked to "speculate" upon her probable life history, and the possible reasons therefor. He was asked to relate to the therapist in detail all that Nelly "said" and to discuss this material fully. Thus, careful guidance by the therapist enabled him to review objectively, critically, and with free understanding a great wealth of both pleasant and unpleasant material, disclosing his relationships to his mother and his comprehension of what he believed to be her understandings of the total situation. Thus he was placed in a situation permitting the development of a new frame of reference at variance with the repressed material of his life experience, but which would permit a reassociation, an elaboration, a reorganization and an integration of his experiential life.

In subsequent therapeutic sessions, a similar procedure was followed, separately, with two other hallucinated figures, "spoken of" by Nelly as her son Henry and his wife Madge, neither of whom the patient could recognize because of his induced amnesia.

The hypnotic session with Henry was greatly prolonged since Henry "told" the patient a great wealth of detailed information which the patient discussed with the therapist freely and easily and with excellent understanding. The patient's interview with the hallucinatory Madge was similarly conducted.

Of tremendous importance in the eventual therapeutic result was the patient's report upon the emotional behavior he "observed" in the hallucinated figures as they related their stories, and his own objective, dispassionate appraisal of "their emotions." He frequently expressed amazement to the therapist and to the hallucinated figures over "their" inability to understand "their own emotions."

To explain this procedure, it must be recognized that all of the material the patient "elicited" from the hallucinatory figures was only the projection of the repressed material of his own experience. Even though a profound repression for all aspects of his problem had been induced, that material still existed and could be projected upon others

since the projection would not necessarily lead to recognition. To illustrate from everyday experience, those personality traits disliked by the self are easily repressed from conscious awareness and are readily recognized in others or projected upon others. Thus, a common mental mechanism was employed to give the patient a view of himself which could be accepted and integrated into his total understandings.

The culminating step in this procedure consisted in having him hallucinate Nelly and Henry together, Madge and Henry, Nelly and Madge, and finally all three together. Additionally, he was induced to develop each of these various hallucinations in a great number of different life settings known from his history to be traumatic, such as a shopping trip with his wife which had resulted in a bitter quarrel over a minor matter, a dinner table scene, and a quarrel between his wife and his mother.

Thus, the patient, as an observant, objective, judicious third person, through the mechanisms of repression and projection, viewed freely, but without recognition, a panorama of his own experiential life, a panorama which permitted the recognition of faults and distortions without the blinding effects of emotional bias.

In the next session, again in a profound somnambulistic trance, he was emphatically instructed to remember clearly in full detail everything he had seen, heard, thought, and speculated upon and appraised critically in relationship to Nelly, Henry and Madge. To this he agreed readily and interestedly. Next, he was told to single out various traumatic incidents and to wonder, at first vaguely, and then with increasing clarity, whether or not a comparable incident had ever occurred in his life. As he did this, he was to have the privilege of remembering any little thing necessary in his own history. Thus, he was actually given indirect instructions to break down by slow degrees the induced amnesia or repression previously established.

The patient began this task slowly, starting with the simple declaration that a cup on the table, in a dinner scene he had hallucinated, very closely resembled one he had had since childhood. He next noted that he and Henry had the same first name, and wondered briefly what Henry's last name was, then hastily observed that Madge and Henry evidently lived in the same town as he did, inasmuch as he had recognized the store in which they quarreled so foolishly. He commented on Nelly's dentures and, with some reluctance, related his fears of dentists, and of losing his teeth, and being forced to put up a "false front." As he continued his remarks, he became more and more revealing. Gradually, he tended to single out the more strongly emotional items, spacing them with intervening comments upon relatively innocuous associations. After more than an hour of this type of behavior, he began to have slips of the tongue which he would immediately detect, become tense, and then, upon reassurance by the therapist, he would continue his task. For example, in comparing Nelly's light brown eyes with Madge's dark brown eyes he made the additional comment, "My wife's eyes are like Madge's." As he concluded his statement, he showed a violent startle reaction and in a tone of intense surprise repeated questioningly, "My wife?" After a moment's hesitation, he remarked to himself "I know I'm married. I have a wife. Her name is Madge. She has brown eyes like Madge. But that is all I know. I can't remember any more—

nothing—nothing!" Then, with an expression of much anxiety and fear, he turned to the therapist and asked pleadingly, "Is there something wrong with me?"

He was immediately reassured that nothing was wrong, that something very good, desirable and right was happening to him, and he was warmly praised for the excellence of the work he was doing, the courage he was showing, and the remarkable clarity of the understandings he was developing. The effect of this reassurance and praise was to make him plunge more deeply and energetically into his task.

Shortly he discovered the similarity between Nelly and his mother, and continued, with excellent understanding, by appraising Nelly as an unhappy neurotic woman deserving normal consideration and affection. This led to the sudden statement, "that applies to my mother too—Good God, Nelly is my mother only I was seeing her for the first time—her eyes are brown—like Madge's. My wife's eyes are brown—her name is Madge—Madge IS my wife."

There followed then a whole series of fragmentary remarks relating to traumatic situations, of which the following are examples:

"The fight at the store—that coat she bought—we almost broke our engagement—birthday cake—shoe string broke—Good God, what can I ever say to her?" After each utterance he seemed to be absorbed in recalling some specific, emotionally charged event in detail. After about twenty minutes of this behavior he leaned forward, cupped his chin in his hands, and lost himself in silent reflection for some minutes, terminating this by asking in a questioning manner, "Nelly, who is Nelly?" but immediately absorbed himself in reflection again. For some time longer, he sat tense and rigid, shifting his gaze rapidly here and there and apparently thinking with great feeling. About fifteen minutes later, he slowly relaxed, and, in a tired voice declared, "That was hard. Henry is me. Now I know what I've been doing, what I've been doing here, and been doing all my life. But I'm not afraid any more. I don't need to be afraid—not any more. It's an awful mess, but I know how to clean it up. And I'm going to make an appointment with the dentist. But it's all got to take a lot of thinking—an awful lot, but I'm ready to do it."

Turning to the therapist he stated, "I'm tired, awful tired."

A series of questions and answers now disclosed that the patient felt satisfied, that he felt comfortable with the rush of new understandings he had experienced, that he knew that he was in a trance, and that he was at a loss to know how to let his conscious mind learn what he now knew in his unconscious. When asked if he wanted some suggestions in that regard, he eagerly indicated that he did.

He was reminded of how the induced amnesia had been broken down by the slow filtering out of ideas and associations by outward projection where he could examine them without fear or prejudice and thus achieve an understanding. With each new understanding he had experienced further reorganization of his experiential life, although he could not sense it at first. This, as he could understand, was a relatively simple, easy task, and involved nothing more than himself and his thinking and feelings. To become consciously aware of his new understandings would involve himself, his thinking, his everyday activities, his own personal relationships, and the interpersonal relationships of other people. This, therefore, would be an infinitely more difficult task. Upon full understanding of this, an agreement was reached to the effect that he would continue to be neurotic in his

everyday life, but, as he did so, he would slowly and gradually develop a full conscious realization of the meaningfulness of his neuroticisms, first of the very minor ones and then, as he bettered his adjustments in minor ways, to progress to the more difficult problems. Thus, bit by bit, he could integrate his unconscious learnings with his conscious behavior in a corrective fashion which would lead to good adjustment.

The above paragraph is but a brief summary of the discussion offered the patient. Although he believed he understood the explanation the first time, as experience has shown repeatedly, it is always necessary to reiterate and to elaborate from many different points of view and to cite likely incidents in which unconscious insights can break through to the conscious before the patient really understands the nature of the task before him. A possible incident was cited for him by which to learn how to let unconscious learning become conscious. On some necessary trip to the store where the quarrel had occurred he would notice some clerk looking amused at something. He would then experience a strong feeling of amusement for no known reason, wonder why, discover that his amusement was tinged with a mild feeling of embarrassment, and suddenly recall the quarrel with his wife in its true proportions, and thus lose his conscious resentments. A few other such incidents were also suggested, and, as subsequently learned, were acted upon. He was then awakened from the trance and dismissed.

The patient's first step to effect a conscious integration, in accord with his trance declaration, was to visit, with much fear, his dentist, thereby discovering, when once in the dental chair, how grossly exaggerated his fears had been. Next, he found himself humming a song while putting on his shirt, instead of examining it compulsively for wrinkles, as had been his previous habit.

Examination of all the family photographs initiated a process of identification of himself, his mother and his wife. He discovered for the first time that he resembled his father strongly and could not understand why he had previously believed so fully that he was the image of his mother. By way of the photographs, he discovered the dissimilarities between his wife and his mother, and that dentures had actually altered his mother's facial appearance.

At first, his adjustments were made singly and in minor matters, but, after a few weeks, larger and larger maladjustments were corrected. Usually, these were corrected without his conscious awareness until sometime later, a measure which had been suggested to him. For example, he had always visited his mother regularly at the hour of her favorite radio program, and he had always insisted on listening to another program which he invariably criticized unfavorably. Unexpectedly, one day, he became aware that, for several weeks, he had been making his visits at a different hour. With much amusement he realized that his mother could now listen to her favorite program, and, at the same time he experienced the development of much insight into the nature of his attitudes toward his mother.

During this period of reintegration, he visited his therapist regularly, usually briefly. Sometimes his purpose was to discuss his progress consciously, sometimes to be hypnotized and given further therapy.

One of his last steps was to discover that he loved his wife and always had, but that he had not dared to know it because he was so convinced in his unconscious that any man who hated his mother so intensely without knowing it should not be

allowed to love another woman. This, he now declared, was utterly unreasonable.

The final step was postponed for approximately six months and was achieved in the following manner.

Walking down the street, he saw a stranger swearing fluently at a receding car that had splashed water on him. He felt unaccountably impelled to ask the stranger why he was swearing in such a futile fashion. The reply received, as reported by the patient, was, "Oh, it don't do no good, but it makes me feel better and, besides, it wasn't the driver's fault, and my swearing won't hurt him."

The patient related that he became obsessed with this incident for several days before he realized that it constituted an answer to the numerous delays in the execution of many half-formulated plans to stage a quarrel with his mother and "have it out with her." He explained further that an actual quarrel was unnecessary, that a full recognition of his unpleasant emotional attitudes toward his mother, with no denial or repression of them, and in the manner of the man in the street, would permit a true determination of his actual feelings toward his mother. This was the course he followed successfully. By following the example set by the stranger, he successfully established good relationships with his mother.

The remarkable parallelism between this final step and the hypnotic procedure of having him project his experiential life upon hallucinatory figures is at once apparent. It illustrates again the value of the hypnotic utilization of the dynamics of everyday behavior.

COMMENTS

In presenting this case material, the purpose has not been to give an understanding of the dynamics involved in the patient's illness nor of the varied nature of his maladjustments. Rather, the purpose of the entire paper is that of demonstrating the values of hypnotic psychotherapy, methods of application, and technics of utilization. A most important consideration in hypnotherapy lies in the intentional utilization, for corrective purposes, of the mental mechanisms or dynamics of human behavior.

Repressions need not necessarily be broken down by sustained effort. Frequently their maintenance is essential for therapeutic progress. The assumption that the unconscious must be made conscious as rapidly as possible often leads merely to the disorderly mingling of confused, unconscious understandings with conscious confusions and, therefore, a retardation of therapeutic progress.

The dissociation of intellectual content from emotional significances often facilitates an understanding of the meaningfulness of both. Hypnosis permits such dissociation when needed, as well as a correction of it.

Projection, rather than being corrected, can be utilized as a therapeutic activity, as has been illustrated above. Similarly, resistances constituting a part of the problem can be utilized by enhancing them and thereby permitting the patient to discover, under guidance, new ways of behavior favorable to recovery. The tendency to phantasy at the expense of action can be employed through hypnosis to create a need for action.

SUMMARY

In brief, there are three highly important considerations in hypnotic psychotherapy that lend themselves to effective therapeutic results. One is the ease and readiness with which the dynamics and forms of the patient's maladjustments can be utilized effectively to achieve the desired therapy.

Secondly, is the unique opportunity that hypnosis offers to work either separately and independently, or, jointly with different aspects of the personality, and thus to establish various nuclei of integration.

Equally important is the value of hypnosis in enabling the patient to re-create and to vivify past experiences free from present conscious influences, and undistorted by his maladjustment, thereby permitting the development of good understandings which lead to therapeutic results.

ELECTROSHOCK THERAPY IN AN OUTPATIENT SETTING

LOUIS LINN, M.D.*

In the few years that have passed since electroshock therapy was first introduced in this country, a vast literature on the subject has grown up. The simplicity of the procedure, its safety in the hands of properly qualified physicians, and the dramatic therapeutic results obtained in selected cases more than justify its extensive clinical use. It is not the purpose of this communication to add one more review to the literature. This has been done so adequately in the past^{1,2} that a similar review on my part would merely repeat what is already known. But this may be said: all evidence thus far based on the statistics of various mental hospitals indicates that the immediate benefits of shock therapy are good, even though the long term results are not so good. Schizophrenics improve but are likely to relapse. Manic depressives recover but are not secure against subsequent attacks.

However, there are certain aspects of electroshock therapy which are not evident in statistical tables. First of all is the effect that shock therapy has had on the general quality of psychiatric work. Compared with the "preshock era," there is at the present time a more intense and widespread interest in the patients of state institutions. Indeed, in many hospitals electroshock therapy has been responsible for the transformation of the individual from the status of inmate to that of patient. Settled routine has changed into active therapeutic interest. Clinical psychology in the state hospital has come of age as a result of the intensive psychological studies carried out on patients before and after treatment. Ward attendants have in many places emerged from their roles as strong-armed helpers to that of medical aides. The wards look different, and the patients look different.

Another aspect of electroshock therapy that is not evident in the statistical tables derives from the heuristic implications of this treatment. One has only to see the extraordinary reorganization of personality which occurs following shock therapy to know that this treatment is both valuable and effective. The results on a deeply depressed patient are largely predictable. In a very high percentage of such cases (70 to 90 per cent) one can bring about restoration of affect to normal levels in a few weeks. Irrespective of whether an episode might have subsided after many months without treatment or that recurrences cannot be prevented, the fact is that electroshock therapy does abort an attack with dramatic suddenness. The mechanism whereby shock

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therapy accomplishes this change awaits elucidation, but our present lack of knowledge does not render the method less empirically useful. The early use of cinchona bark in the treatment of malaria was rational and empirical even though nothing was known of the plasmodium or of quinine.

A third, and very important, aspect of electroshock therapy to which I wish to call attention also is not found in statistical tables. I refer to its benefits in the treatment of patients outside mental hospitals. This point, the value of shock therapy as an outpatient procedure, will constitute, in the main, the subject of this paper. The benefits of home, office and outpatient treatment may be illustrated by a few representative case histories.

ILLUSTRATIVE CASE HISTORIES

CASE I.—R. P., a 50 year old white man, had hypochondriacal reactions for many years. Early in December 1945 he became increasingly tense, anxious and sleepless. He was treated with sedation and superficial psychotherapy without relief, and on December 20, 1945 he became frankly psychotic. He became very panicky, responded to auditory hallucinations, gestured bizarrely, and could not sleep. He expressed the fear that a gang was on its way to his house to destroy him. Despite his fears and suspicions, his wife was able to persuade him to submit to electroshock therapy. On December 23 he received his first grand mal seizure, the treatment being administered in his own home. The next day he was somewhat quieter and more receptive to treatment although still actively psychotic. Two days later he was given a third treatment. Following this he returned to his prepsychotic state. He went back to work on January 2, 1946, resumed all his former duties, and has performed satisfactorily since then. He received six more treatments at weekly intervals in January and February, 1946. At the present writing, nineteen months after the acute psychotic episode, he is in complete remission.

Comment.—Ordinarily, a patient in such a disturbed psychotic state would have been hospitalized. Most likely he would have been observed for several weeks before treatment was instituted. Following the remission he would have been kept in the hospital, for further observation, for several more weeks. On his return he would have borne the lay "stigma" of having been in a mental hospital. His associates would have hesitated to let him resume his responsibilities. The altered relationship with his colleagues would undoubtedly have created psychological hardships for him during his convalescent period. An important item might have been the expense of medical and nursing care in a private sanitarium. As a result of the prompt administration of electroshock therapy at home he did not leave his family. Fortunately, his illness coincided with the Christmas vacation. When he returned to work on January 2 his business associates knew nothing about his mental illness, and their relationship to him was unaltered.

The possibility that the patient would have recovered without electroshock therapy and that he may have a relapse in the future is not relevant at this time. What might have been a catastrophic dislocation of an entire life was converted into a minor incident. In addi-

tion, the rapidity with which the psychosis was terminated and the effective contact with the environment which the patient was able to maintain during his convalescence are of great importance in psychological rehabilitation. Here is an aspect of shock therapy which cannot be gleaned from statistical tables alone.

Perhaps an even more dramatic demonstration of the importance of electroshock therapy as an outpatient procedure is seen in the following case.

CASE II.—H. K., a 47 year old white man, taught art in a high school for about twenty years. He had a cyclothymic personality, characterized by episodes of mild depression alternating with periods of elation. However, these mood swings were not incapacitating until 1940. In 1939 the patient married. According to his report the marriage was fairly congenial. With the onset of the severe depression in 1940, he had to discontinue his teaching. After many months of inactivity and despondency he was admitted to a hospital where he received injections of insulin in subshock doses. This did not alter the course of his illness. His depression continued, and after several weeks in the hospital he attempted suicide by slashing his wrists. He was transferred to a psychiatric hospital where he received a course of electroshock therapy. After eight treatments he was discharged from the hospital in remission. A few months later he returned to his teaching post. Altogether he lost about a year and a half from his school work. His wife responded with poor understanding to his illness, as a result of which she left him.

The patient taught successfully up to May 1946 when his depression returned. For a week he attempted to continue his school work, then he became increasingly depressed and sleepless, cried, and entertained thoughts of suicide. He was panic-stricken at the thought that another nightmarish year and a half lay before him. On Wednesday, May 2, he received his first electroshock therapy. By May 11 he had received a total of five treatments. On May 13 he returned to school in full remission, having lost only a week from his work. In December 1946 he experienced a return of his depression. Two treatments aborted this episode with a loss of only three days from his school work. His work has continued on a high artistic level.

Comment.—Compare the minor disturbances in the life of this man when his depression was treated on an outpatient basis with the results of hospitalization in 1940. One may speculate what would have happened if his previous episode had been interrupted as simply as the more recent ones. His marriage might have been preserved and he might have been spared the traumatic experience of a suicide attempt.

One of the most serious consequences of mental illness is the frequent disruption of the home, especially when a mother of small children has to be taken to a hospital. Repeatedly, it has been our good fortune to be able to preserve homes by means of electroshock therapy. The following are typical case histories.

CASE III.—R. U., a 27 year old white woman, the mother of a girl of 4, was a cheerful, outgoing individual, congenially married and apparently well adjusted until January 1946 when she became depressed and retarded in her speech and actions. She lost interest in her husband and child, stopped eating, became sleepless, expressed feelings of unreality, had uncontrollable crying spells, and expressed suicidal ideas. She continued in this condition until June 20, 1946 at which time she was referred for electroshock therapy. By July 18 she had received eight treat-

ments, all grand mal seizures. She went into a full remission and resumed her household duties. Her husband reported that she is her old cheerful self.

CASE IV.—M. K., a 32 year old white woman, had a depression, several years before, which subsided without treatment after a number of months. Her present depression started in September 1946, shortly after the birth of a daughter. It was typical in all respects. On December 3 she received her first treatment. By January 16, 1947, after she had received nine treatments, she went into complete remission and resumed her household duties. She has been seen in psychotherapeutic sessions at weekly intervals in an endeavor to work out some of her dissatisfactions with the role of housewife. However, she is normally cheerful and outgoing in her manner and leads a satisfactory social life.

CASE V.—M. C., a 32 year old white woman, with one daughter of 5, was apparently well until January 1946, shortly after the return of her husband from military duty. She became depressed and confused, had crying spells and expressed feelings of hopelessness and personal unworthiness. Within one month she was completely incapacitated by her depression and was practically mute. She received her first treatment on February 20 and by March 8 had her sixth and last treatment. Now she was able to communicate freely. During subsequent psychotherapeutic sessions the fact was elicited that her sexual relations were unsatisfactory. Consultation with the husband revealed that they were both very much in need of elementary instruction. Appropriate guidance led to satisfactory sexual adjustment. The patient is apparently in full remission.

CASE VI.—B. G., a 29 year old white woman, with two girls, ages 4 and 8, was apparently well until October 1946 when she went into a typical depression. On December 18, 1946 she received her first shock treatment and on February 4 the ninth and last. By that time there was complete remission and she was taking care of her children and her home. She was seen at weekly intervals for about two months thereafter. It was found that the patient was having difficulties with an overdemanding, aging mother. She was guided in adjusting to this matter. The patient is in a good remission at this time.

CASE VII.—C. F., a 35 year old white woman with two children, ages 3 and 9. was well until May 1946. Following a series of financial reverses her husband had a "nervous breakdown" in January 1946, which lasted three months, during which he was nursed by the patient. As he recovered from his illness the patient became moody and withdrawn. She lost interest in her children, became suspicious of those about her, accused her husband of wanting to harm her, reacted to auditory hallucinations, spoke incoherently, and smiled inappropriately. She was admitted to a hospital where she received seven electroshock treatments. She apparently went into remission. Against the advice of the physicians the family removed her from the hospital. She relapsed almost as soon as she got home. She became more violently antagonistic toward her husband, developed the idea that an attempt had been made to kill her at the hospital, and refused to return for further treatment. However, she was willing to accept treatment as long as she could stay with her parents. Between December 4 and March 17, 1946 she received a total of sixteen treatments. She went into remission after the sixth treatment, and remained well thereafter. She is back home with her husband and children.

Comment.—In each case the mother was unable to care for her children. The guilt feelings concerning the neglect of the children, which were prominent in the clinical picture, were promptly relieved by treatment. Rapid restoration to normal promoted sympathetic understanding on the part of the husband. One must also regard electroshock

therapy in these cases as a mental hygiene measure in the prophylaxis of emotional disturbances in the children of a psychotic mother.

The question why one acute psychosis is followed by recovery and another becomes chronic cannot always be answered with assurance. From a statistical study of results obtained with electroshock therapy we know that the earlier the treatment is administered in the course of the illness the better are the results. That is to say, the shorter the period of alienation from reality, the higher the percentage of recovery. Hospitalization invariably results in a delay in the initiation of treatment. Furthermore, the strange surroundings and activities which characterize the ordinary mental hospital undoubtedly serve to emphasize and exaggerate the break with reality which so frequently characterizes the onset of grave mental illness. Preservation of contact with loved ones, with familiar surroundings, with well known routines and activities must exert a beneficial effect on the course of a mental illness. In younger age groups, where habits and concepts are still in the process of formation, these factors assume particular importance.

CASE VIII—S. K., a 17 year old white male, was always irritable and nervous and a bedwetter until the age of 10. He finished high school satisfactorily at the age of 16. He was normally sociable with young men of his own age group and he went out with girls occasionally. He participated in high school and college athletics. He was a prize winning gymnast. He entered college in September 1945, but complained of difficulty in concentrating on his studies. In his second term his difficulties increased. He withdrew from all his friends, and developed crying spells. He had a herpes simplex on his lip, which he was sure was a syphilis lesion. He expressed feelings of personal unworthiness, stating he was "no good" and "too dumb to live." He refused to get out of bed in the morning, stayed in the bathroom for hours, and quit school.

Electroshock therapy was started on January 10, 1946. The patient received a course of twelve treatments, which was concluded on March 18. After the sixth treatment in February he improved sufficiently to return to school, though the treatments were continued. A relatively light program of studies was arranged for the patient. He passed his courses successfully, made up some of his lost work that summer and resumed his regular program of second year studies in the fall of 1946.

CASE IX—V. G., a 16 year old white boy, a student now completing the third year of high school, was well until April 1947, at which time he went into a panic characterized by the delusion that Nazis were outside his house, trying to get in to destroy him. He became sleepless, suspicious of those about him and refused to take food or drink. Weeks of tension due to much study, working late as an usher in a theater and nursing an ailing mother culminated in his illness. He did not permit members of his family to leave the house and assaulted visitors. He was in a violent overactive condition when he came for the first treatment, but with the assistance of members of the family he was given intravenous pentothal and a grand mal seizure was induced. When he came out of it he resumed his former hostile, suspicious manner. Guided by Army experience in the control of acute psychotic excitements,⁴ a second convulsion was induced one hour after the first. He became quiet and tractable. Twenty-four hours later he was given his third treatment. Now he was mute and inaccessible but no longer negativistic or violently assaultive. Following two more treatments at two day intervals he went into a spectacular remission. Three additional treatments at weekly intervals and several therapeutic sessions led to resumption of his social contacts and return to school.

Comment.—The clinical impression in both cases justified the diagnosis of incipient, chronic schizophrenia. Both patients were no longer manageable at home. Electroshock therapy made it possible for them to remain with their families and brought about prompt recovery. Obviously one cannot draw final conclusions from a few cases, but it is equally clear that outpatient shock therapy of young schizophrenics can be highly effective. Perhaps most important is the fact that they are kept out of institutions. Successful shock therapy makes possible the application of a rational program of psychotherapy.

Psychiatric problems in the older age groups are also capable of leaving wreckage in their wake. It is a common observation that old persons do not tolerate hospitalization well. Of 638 senile patients admitted to a better than average state hospital ⁴ 47 per cent were dead at the end of one year. Fifty-eight per cent were dead in two years. In spite of this death rate senile patients occupy a huge percentage of all the beds in state hospitals. Most of them are regarded as hopelessly ill and little is done aside from custodial care. Electroshock therapy has been of benefit in a number of selected cases. Elderly patients were found to tolerate it as well as younger ones,⁵ and many reactions that had been dismissed as hopeless, the result of irreversible changes in the brain, showed spectacular improvement following treatment. The following cases exemplify what electroshock therapy has to offer some of those previously designated "hopeless" cases.

CASE X.—A. H., a 60 year old white woman, gave a history of hypochondriacal tendencies most of her life. She has two sons and a daughter, happily married. After the death of her husband in 1937 she lived in a small apartment by herself, making an apparently adequate adjustment until March 1947 when she became depressed and sleepless, had episodes of uncontrollable weeping and expressed suicidal ideas. A diagnosis of cerebral arteriosclerosis with early senile changes was made and institutionalization recommended. After wrestling with the problem at home for a long time the family accepted the advice to give ambulatory shock treatment. Eight treatments were administered over a period of six weeks. The depression disappeared after the sixth treatment. Though the patient still has her old hypochondriacal tendencies, they are less marked than they have been for years. She is back in her apartment, fully capable of taking care of herself and apparently quite happy, and so is the family.

CASE XI.—M. H., a 62 year old white woman, happily married, with three children with families of their own, was well until February 1947. Apparently precipitated by a housing problem, she became sleepless, agitated and suicidal and lost 40 pounds. Custodial care was advised by a physician, but on further thought it was decided to try electroshock therapy. She received seven treatments over a period of eight weeks. She went into full remission after the fifth treatment. She has regained her 40 pounds. The housing difficulty was straightened out and life within the family has been restored to its previous tranquillity.

Comment.—True depressions characterized by agitation, weeping and suicidal ideas, occur in patients with a well preserved intellect, while senile deterioration is associated with apathy or euphoria and not depression. So that depression in an elderly patient may occur in

the presence of a well preserved intellect and calls for electroshock therapy. These two cases were selected because in both of them the diagnosis of cerebral arteriosclerosis was made and institutional care recommended.

SPECIAL CONSIDERATIONS

Method of Administration of Outpatient Electroshock Therapy.—This is not the place to go into the details of electroshock therapy technique but a word may be said to show that the treatment is not too complicated or hazardous and that it can be administered in an outpatient setting by a properly qualified psychiatrist. The patient and family are told that the treatment consists of a brief period of sleep, induced in a special manner. To avoid anxiety no mention is made of electricity, shocks or convulsions. Food is withheld for a minimum of six hours preceding treatment. The patient is first instructed to void, and to discard glasses and all removable artificial dentures. He is then put to sleep with an intravenous injection of sodium pentothal. *That is all that the patient knows of the treatment.*

After the patient is asleep, the usual precautions are taken to prevent injuries, the electrodes are applied to the skull, and a grand mal seizure is induced. Thanks to the pentothal, the patient will almost invariably sleep quietly for about fifteen to thirty minutes after the convulsion has occurred. In this way he sleeps through the initial period of confusion which occurs following the convulsion and is spared considerable anxiety. For further reassurance he is permitted to awaken spontaneously in the presence of a member of the family. At this time, it is generally possible to establish good transference which is of inestimable value in subsequent psychotherapy. After about an hour the patient is permitted to go home. Aspirin may be given for headache and seconal for sleeplessness, otherwise no special instructions are necessary.

Indications for Outpatient Electroshock Therapy.—The two least controversial indications for electroshock therapy are mental reactions characterized primarily by severe depression, and acute schizophrenic reactions. If the patient is able to establish some rapport with the physician and discuss his problems and if in the case of depressions there is no serious danger of suicide or, in the case of the schizophrenic, he is manageable at home, there may be a few days of exploratory psychotherapy before deciding on the proper course of treatment.

The Number and Frequency of Treatments.—Most patients receive two treatments a week until they go into remission, which generally occurs between the fifth and eighth treatments. In the case of a depression, the patient receives two more treatments, a week apart, after remission has occurred. Schizophrenics receive about six weekly treatments after remission has occurred. In the suicidal patient, or the disturbed schizophrenic, treatments are given more frequently in the

beginning. Where the patient is young, and is in good physical condition, as many as three treatments can be given in the first twenty-four hours³ in an endeavor to bring about a level of ego integration compatible with keeping the patient at home.

Psychotherapy and Outpatient Electroshock Therapy.—It has been brought out repeatedly in the foregoing case histories that outpatient treatment lays a solid foundation for psychotherapy. In 1939 Meyer-son⁶ reported on a method of treatment of schizophrenic patients in mental hospitals which he called "Total Push." The idea of this treatment was to expose the psychotic patient to a program of sustained environmental pressure during which he is subjected to interesting stimuli to elicit responses from him almost in spite of himself. This forced reality contact was effective in a high percentage of cases in bringing about improvement. Treating the patient in an outpatient setting provides opportunity for total push to a degree rarely attainable within a hospital. Kept in contact with his or her responsibilities, whether they be in school, on the job or in the house, the patient experiences a powerful push toward reality. In addition, continued contact with family and friends makes possible a variety of social activities which may elicit effective responses from the patient.

From a psychoanalytic standpoint, the task of treatment of many psychotic reactions consists of a strengthening of the patient's ego,⁷ so that it can deal more effectively with reality. This strengthening is accomplished most effectively by means of affection, which parents and other near and dear relatives can best supply. Thus the psychotherapist may point out to an undemonstrative parent or spouse the need of giving more affection, he may be instrumental in relieving a patient of excessive loads of responsibility, or help to smooth over conflicts resulting from the demands of an overattached mother and the duties of marital life.

Finally comes the direct psychotherapeutic contributions which the physician himself can make. A patient who improves following electroshock therapy is a grateful person and readily makes a strong positive transference, which can be used very effectively. Most patients who recover from a psychotic episode following shock therapy generally are reluctant to probe into the psychodynamics of their illness. Simple guidance, counsel, explanation, reassurance and the like strengthen the positive transference and make the patient feel that in the physician he has a good friend in time of need. As a rule only a small group of patients want to explore more deeply the reasons for their breakdown or are able to cooperate. Such individuals can derive considerable benefit from exploration of unconscious material. However, in those patients who have recovered from a psychosis it is particularly important not to press for material.

Contraindications to Treatment.—Only two conditions, namely, heart failure and acute pulmonary disease, are absolute contraindica-

tions. In spite of the dramatic appearance of a convulsion and the apparent violent exertions involved, patients who are malnourished, enfeebled, aged or otherwise debilitated tolerate convulsions remarkably well. Moore⁹ reports on the treatment of over 2000 patients with cardiovascular disease with and without hypertension, pulmonary tuberculosis, skeletal disorders, cerebral and generalized arteriosclerosis, advanced age, and syphilitic meningoencephalitis. No complications of moment, except in one patient with known severe cardiac disease who died one week after treatment was finished, were encountered. Hypertension not only is not a contraindication to electroshock therapy but is often benefited by the treatment since the hypertension itself occasionally is a somatic expression of the psychic disorder being treated. However, the paucity of contraindications does not absolve the psychiatrist from the responsibility of confining the treatment to properly indicated psychiatric categories, since the treatment is not without danger.

Complications and Sequelae.—Deaths, though rare, have been reported following shock therapy, and the reason is not always clear.⁹ The danger of death by suicide or from exhaustion is much greater than is the danger from electroshock therapy itself. Fractures of various bones used to be fairly common; now they are very rare. Compression fractures of the vertebrae do occur but they usually are asymptomatic, are associated with no orthopedic or neurologic manifestations and generally require no treatment.¹⁰ Occasionally a patient complains of headaches or muscular soreness following treatment. These are readily relieved by aspirin. The overwhelming majority of the patients go through the treatments without any complaints whatever.

An annoying complication and sequel to electroshock therapy is memory defect, most likely the result of a certain amount of brain damage. This is inferred from the electroencephalographic abnormalities following electroshock therapy and from characteristic psychological changes. However, if the total number of treatments is kept down to a minimum, if the treatments are spaced as far apart as is practical, and if the current used to produce the convulsion is kept down to the least necessary amount, then these organic changes are minimal. In any case, they are not permanent. Depending on the number of treatments, improvement in memory and in the electroencephalographic findings occurs rapidly. It takes from one to six months before evidence of organic impairment disappears completely.

REMARKS

It has been frequently stated that electroshock therapy should be administered only to hospitalized patients. It is felt by some that such therapy in an outpatient setting exposes the patient to unwarranted hazards. From the foregoing summary of the technic of treatment, the indications and contraindications, the possible complications and

sequelae, it is clear that electroshock therapy is not to be embarked upon lightly. The decision whether or not to proceed with treatment often demands considerable experience and fine clinical judgment. But this only means that electroshock therapy should be administered by a properly qualified psychiatrist. That it can be abused is true, but the same holds for other branches of medicine.

The only relevant question is this: Given a patient with a mental illness for which shock therapy is indicated, will treatment as an outpatient be a greater hazard than treatment in a hospital? The unequivocal answer is that the danger is not greater. As a matter of fact, electroshock therapy lends itself particularly well to office practice. The precautions are the same, the complications are no more frequent, and the safety is no less in a properly equipped place. On the other hand, the gain from staying out of a hospital can be great indeed. The cost of private hospitals is almost prohibitive for most people and state hospitals cannot readily give the treatment to all who need it or could profit from it. Aside from the environmental and love privation away from home, the delay can be costly in suffering and in the possibility of recovery.

Decidedly, electroshock therapy should not be restricted to patients in hospitals. On the contrary, its use on an outpatient basis should be expanded so as to make it useful to infinitely more patients and to lessen overcrowding in mental hospitals. While controlled studies comparing the efficacy of electroshock therapy in and outside hospitals have not been carried out on a large scale, the few published data available indicate clearly the superiority of the results achieved with outpatients compared to a similar group of patients treated by the same people and the same technic within a mental hospital.¹¹ Ambulatory treatment opens a rich opportunity for psychotherapy, for environmental manipulation, and for reeducation of the patient's family. If it can accomplish these objectives outpatient electroshock therapy becomes a great contribution to psychiatry.

SUMMARY

Electroshock therapy of selected patients in an outpatient setting frequently obviates the need of hospitalization in many cases. Keeping a patient out of mental institutions accomplishes several things: he is kept at home in familiar surroundings where he can be nursed by loved ones, and spared the possible trauma of sojourn in a strange environment; he may be able to maintain moderately effective adjustment at work, at school, as a housewife or as a parent, throughout the course of treatment. These factors are of great psychotherapeutic importance. Furthermore, outpatient electroshock therapy makes it possible for the psychiatrist to continue active psychotherapy on as intensive a basis as he chooses. Finally, it becomes possible to enlist social service agencies in a program of reeducation of the family and

to make beneficial changes in the patient's environment to a greater degree than when the patient is institutionalized

As a precaution, electroshock therapy should be administered by a properly qualified psychiatrist and on an outpatient basis only where clearcut indications for its use exist. These are: (1) clinical pictures characterized predominantly by depression and (2) acute schizophrenic reactions.

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ANXIETY STATES: THEIR RECOGNITION AND MANAGEMENT

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ANXIETY is an emotional response of unpleasant character which portends danger or threat to the individual. The emotion is usually diffuse, without specific focus, and is generally less intense in nature than fear, which is more topically focused in terms of fear of—something. Every human being, even the normal, experiences anxiety, particularly in situations which constitute recognized threats. In our definition, we are concerned, however, with abnormal or pathological states of anxiety severe enough to constitute a disability, and usually rendering the individual ineffectual or incompetent at the usual tasks of life. Such pathological anxiety may occur in acute, overwhelming attacks lasting a few minutes to a few hours, usually subsiding or passing off; or it may be protracted, chronic, lasting for weeks, months or years at varying levels of intensity. More often than not it occurs without adequate precipitating cause, appears to come out of the blue, and its true causes are rarely understood by the sufferer.

RECOGNITION OF THE ANXIETY STATES

The Clinical Entity of Anxiety.—The picture presented by the individual caught in such pathological anxiety is well known to every practitioner. The sufferer may be observed to be uneasy, flushed, restless; he may perspire freely and present wet palms or moist axillae; his pupils may be dilated, his hands and body tremulous, his breathing rapid and shallow, his pulse accelerated, his tongue and mouth dry. He may suffer polyuria or diarrhea, may be nauseated and vomit, and may have total loss of appetite. He may complain of insomnia and frightening dreams, of constriction in his chest, inability to swallow, or difficulty in taking a deep breath, or he may suffer with cardiac pain and a sense of impending death. In brief, his total organism may be involved at the cardiorespiratory, vascular or neuromuscular level. He is usually in the grip of a profound autonomic upheaval, and upon further medical study he may be shown to be suffering from elevated blood pressure, increased leukocyte count, raised blood sugar level, sometimes hypoglycemic response, hyperventilation tetany, and other metabolic and chemical imbalances. At the very moment of experiencing these sensations, he may recognize that there is nothing in reality of which to be afraid, although he is very apt, upon questioning, to reveal that he is afraid of dying. Instead of being mild and discrete

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and reversible phenomena, states of anxiety may become protracted, chronic and sweepingly disabling. They may attain the status of panic, and may arise whenever external stress or threat is present, or, more commonly, whenever the threat derives from unrecognized conflicts within the person. These states exemplify the pathology of anxiety. Other significant effects are known. Experiments have shown that an adrenalin-like substance in the blood of patients suffering from anxiety is capable of altering the smooth muscle function of the isolated rabbit intestinal strip. Psychological experiments have revealed the profound influence of anxiety on the thinking process, on the keenness of concentration, and on the functions of retention, active and passive attention, and abstract thinking.

Anxiety Accompanying Other Psychoneurotic States.—Anxiety of course does not occur always as an isolated entity, for some degree of anxiety occurs with many of the other psychoneurotic reactions. It is a frequent accompaniment of depressive states, and a characteristic finding in involutional melancholia, and it often underlies and is evident in much of the pathology of schizophrenia. It is the operating link in most of the psychosomatic manifestations. It is a common accompaniment of physical disease and infection. It is apt to be present to some degree in all convalescent patients, and, when present, it may result in delayed convalescence, protracted symptoms, and failure to respond to the usual medical regimen. Anxiety may be the fundamental cause, may aggravate, may explain fluctuations, or may determine the entire course of multiple physical illnesses (particularly the so-called psychosomatic disorders). There is growing evidence to believe that anxiety may usher in such conditions, and that protracted states of anxiety may ultimately lead to permanent and irreversible chronic changes of blood vessels, mucous membranes, skin or joints. Wherever autonomic innervation prevails, one can expect anxiety to manifest itself at one or another end organ. Anxious people are often chronically fatigued people. They frequently overeat, and become obese. They may reject food, and develop anorexia. They may suffer reversal of the intestinal function, with either constipation or diarrhea as results. Such individuals may recognize their anxiety for what it really is; they may have partial insight into the accompanying or aggravating role of anxiety; or they may be totally without insight as to the essential emotional nature of their condition.

Anxiety as a Defense Mechanism.—Today we know that anxiety is the natural defense mechanism of the human being which is called into play whenever the person is threatened, whether from without or from forces within the personality. It serves as a medium for the mobilization of defenses. As Cannon postulated, it is a homeostatic mechanism devised to prepare the threatened organism for flight or fight. Such an individual, overgalvanized for response, unable in our society to resort either to flight or fight, blocked in any direct mode

of release or expression, succumbs to the full fury of the overmobilized state with some or all of the cardinal symptoms already enumerated.

Psychopathology of Anxiety.—Anxiety is best understood in terms of its psychological significance to the individual. Older theories which dealt with constitutional liabilities advance us none in understanding. It is time for recognition of the fact that the pathologically anxious adult has almost invariably been an anxious child. However, the attempts to interpret the so-called neuropathic traits of childhood (nail-biting, enuresis, nightmares, temper tantrums and allergic responses) as arguments for an inherited constitutional or hereditary liability prove little. They only argue for the early and fundamental insecurity of individuals at that age period, with its possibilities for easy reactivation in later life. Those earlier investigators who laid stress on the family history as being of particular etiologic significance have produced no evidence other than that parents or relatives are commonly anxious or neurotic, and that this clearly operates as a significant environmental pattern.

The purely psychogenic explanations are the most current and the most revealing. There can be no real understanding of anxiety which does not recognize that emotional conflicts lie behind it, that these conflicts are beyond the average patient's capacity to resolve alone, that the true causes are largely unconscious or unknown to the patient, and that the manifestations are never conscious, or feigned, or deliberate.

Normally we expect to feel anxious when threatened with injury, or have an accident, when as students we are faced with a difficult examination, or when as adults we are beset with economic or other problems, or are threatened by disaster or war. For many doctors the anxiety which attends physical disease may be easier to understand. Consider the emotional reaction of the average man to an attack of coronary thrombosis. At such a time every individual is rendered less mature, more dependent, more in need of support and comfort. These emotions often go unrecognized in the treatment of individuals following ordinary infections, operations and the like. In almost everyone, during convalescence the anxiety abates, the personality gradually reassembles itself toward the mature, self-directing person it was, the childlike need for comfort and protection is once more abandoned, and natural recovery takes place with the disappearance of anxiety. Some such degree of anxiety is also commonly present in chronic disease, and it may play a role in rendering the chronic patient overly submissive and dependent, undesirous of giving up the protected role, unwilling to face mature responsibility, dependent upon the secondary gain of illness, hospitalization or compensation. Without due recognition of this fact, bed rest is often needlessly and dangerously prolonged, or the converse occurs in which the individual is ruthlessly urged to activity long before the anxiety abatement makes it possible for him to comply. Thus in every sick patient the factor

of anxiety must be recognized. This fact has long been known, and is evidenced in the fact that a high incidence of emotional disturbances (27 per cent at the University of Chicago Hospital, 30 per cent at the New York Hospital) is present in patients admitted to general hospitals for conditions not recognized as primarily emotional in origin.

Aside from these everyday problems of medical practice, we recognize "anxiety states" occurring as reasonably distinct clinical entities (psychoneurotic in nature). In these conditions the anxiety is perceived and experienced as the main complaint. The response is diffuse (in technical language, free-floating), is rarely attached to situations or objects, and is not warded off by any of the common psychological mechanisms of defense. It thus contrasts itself with those psychoneurotic reactions in which anxiety is primarily manifested in heightened and frightening concern about the health, or about specific organs (commonly called hypochondriacal reactions) in which there may be partial or total suppression of the anxiety sensations. It differs also from those states in which the anxiety is expressed as overwhelming exhaustion and diffuse nervousness (neurasthenic reactions), or where the anxiety is displaced upon some specific object, action or situation (phobias, obsessions, compulsions).

Let us view anxiety, therefore, as serving a warning or threat to the person. In such individuals who suffer anxiety states we are dealing with persons whose personality equilibria are easily upset, who already have a strong preparedness for anxiety, and in whom external pressures usually act as triggers to set off reservoirs of anxiety potentials which have been present, though unfelt, until the pathological reaction is precipitated. In other words, a reasonably well-compensated personality may rather suddenly decompensate.

Anxiety Responses.—No individual can go through life without some degree of anxiety. The mechanisms developed and employed by the human being to deal with anxiety are multiple. The most common is that of *repression*. The individual denies or forces out of consciousness the external or internal threats which might overwhelm him or give rise to undue anxiety, against the pain of which he tries to protect himself. Normally this mechanism operates successfully in all of us. In others, the mechanism may break down. Then it results in devious and substitutive responses and reactions. Another common mechanism is that of *regression*. Through this the individual retreats to earlier patterns of coping with stressful situations, sometimes resorting to strangely infantile and childlike habits of response. Thirdly, the anxious person may *identify* himself with someone stronger, a substitute parent figure (very commonly the doctor or nurse), and find support in such identification. Fourthly, he may utilize the mechanism of *denial* of anxiety such as one commonly sees in the tuberculous or arthritic patient, with his inability to face the serious import of his disorder. *Conversion* results in the displacement of anxiety to other

bodily manifestations such as hysterical symptoms, with their characteristic emotional blandness and protection from the pain of anxiety. Anxiety may be partially *displaced* from its true source and experienced as phobias (a condition wherein the true source of anxiety is hidden, and a meaningless object becomes the topical focus of the anxiety experience). Anxiety may be *projected* in terms of blaming others, or in seeing in others the attributes or conflicts which the patient cannot face in himself. The common 'sour grapes' attitude is an attempt at a *devaluation* of a true anxiety producing situation. In many individuals the mechanism of *reaction formation* is a characteristic solution. This refers to the development of attributes of character which are in direct opposition to the fundamental strivings of the individual, as exemplified by the extremely passive person whose submissiveness covers up his unrecognized fundamental aggression and hatred. Finally, both the well and the psychoneurotic individual commonly utilize the mechanism of *sublimation*, which in essence is the channeling of anxious emotions into patterns of social contribution and productivity.

These are the common psychological means by which every individual tries to ward off the anxiety experience. Other mechanisms have already been hinted at, the channeling of anxiety into specific organs (the somatization reaction), the development of specific psychoneurotic reaction types, the overwhelming of the organism by anxiety to the point of suicidal depression, the total retreat of the personality before anxiety to levels of schizophrenic disorganization.

In studying the anxiety response in any given patient, one commonly finds upon inquiry that immediate environmental disturbances have been present at the onset of the anxiety response. These may be overwhelming and catastrophic (as in war combat experiences), or they may be relatively trivial in susceptible individuals. Many factors may operate as precipitating or trigger stimuli. These may be purely physical such as poor nutrition, physical exhaustion, or accident, or they may be largely emotional factors operating at home in domestic difficulties, sexual incompatibilities or frustrations, financial worries, thwarted ambition, discouragement, fright, or lack of sufficient outlets for recreation or relaxation. Looking further however, one sees that these are usually predisposed individuals in whom some such experience merely sets off or reactivates earlier conflicts or emotional insecurities. One always finds that early patterns of behavior and childhood reactions are being drawn upon in the clinical anxiety states, and that, faced with great anxiety, the individual characteristically retreats to earlier patterns of security or habits of meeting difficulties.

The basic nature of the disrupting conflicts has been best explained by Freud and his disciples as follows. Primitive and powerful unrecognized urges, desires and instincts (technically called the id) seek always for expression in everyone. During infancy gratification is direct

and uninhibited, and concerns itself largely with certain pleasurable sensations (oral, anal and genital), and with the need to be loved. As the child grows older, these primitive needs are increasingly prevented from gratification through the forces of his acquired conscience, ethics, and mores which he takes over directly from his parents because of his need to identify increasingly with them. He comes to fear loss of love or punishment for continuation of these satisfactions. He acquires a conscience (the superego). These drives and conflicts exist in spite of the character features which the individual habitually displays (the ego), which comes to play the role of mediator. In psychoanalytic concepts these basic conflicts are always unconscious. They persist throughout life (although rarely recognized in their infantile forms). The impulses retain their high initial emotional charge. They seek for perpetual gratification, and when denied expression or awareness by the superego, seek for expression in dissociated, disguised or substitutive fashion. It is this ceaseless and fundamental internal conflict that generates anxiety. While all individuals carry some of these conflicts within them, the normal person is able to sublimate them into healthy channels. Others repress them completely; some suppress them only partially; and in others the forces of repression break down completely, and the individual is overwhelmed.

We know from war experience that every individual has a breaking point. For some very stable people, great external stress is necessary to cause a personality decompensation. In others, very little stress is required to produce anxiety. Why do some react with anxiety and others not, under roughly comparable circumstances of strain? The answer lies in what we have just discussed; some people are more predisposed because of the degree of internal conflict which characterizes their personalities. Where great internal conflict exists, there is relatively little resistance to external stresses. To understand and to treat these disorders, both the external and internal forces must be studied and understood.

TREATMENT

Faced with such a patient, the physician's first desire is to bring some immediate relief. He can usually do it symptomatically and promptly with *sedatives* but, unless the fundamental causes are eliminated, this is a bad way of providing relief, for it may lead to dependence or even addiction. Other physiological approaches are known: *subcoma insulin therapy* given in a hospital offers valuable relief. There are certain chemicals in the offing, like dibenamine, which may have specific action on anxiety. Techniques like *narcosynthesis*, which utilizes intravenous sodium amytal or sodium pentothal for the quick release of significant memories or emotional content, belong in the hands of the psychiatric specialist. The manner in which the doctor handles such a patient will be more decisive than any other factor either to relieve or to entrench the anxiety response.

The patient wants the assurance of a thorough *physical examination*, and this should be done promptly, and not repeated too often. In the process, the physician must control his facial expression, he must appear confident, competent and reassuring. He should not linger too long over the apex with his stethoscope. The authoritative statement of his negative physical findings should be presented calmly. If he has doubts as to minor irregularities, he should keep them to himself for later evaluation. He should not hint at a functional murmur, or a mild elevation of blood pressure. He does well to formulate his negative findings to another member of the patient's family in the patient's hearing. Until this procedure is taken, he cannot move forward in therapy.

The physician should then formulate the reaction for what it is. He should make it clear that he wants to go forward in getting at the causes by further exploration, then or at a later date, of the patient's personality and sources of strain or anxiety. If he gives sedation for an emergency, it should be formulated as such, and only to tide the patient over until the necessary explorations can be made. He should reassure the patient that he will stand by whenever needed until the problem is solved.

Psychotherapy.—If the physician has no intention of undertaking psychotherapy, which is the only valid therapy for these conditions, he should refer the patient, after proper preparation, to a psychiatrist for consultation or therapy.

What then can the general physician who wants to undertake psychotherapy do in the treatment of anxiety states? He must recognize that psychotherapy is his only effective tool and he may feel he knows very little about it. He must begin with the conviction that medicine deals not primarily with disease entities or organ pathology but with human beings reacting to various kinds of noxious stimuli. His greatest effectiveness lies in taking a complete and proper history which gives due emphasis to the individual's emotional make-up, background and prevailing patterns of response. He must learn how to establish an effective rapport or working relationship with his patient and he will succeed in this in direct proportion to the degree of genuine interest he shows. This is facilitated by his sensitive and kindly inquiry into the patient's personal life, his respect for the facts he obtains thereby, his genuine unfeigned desire to be helpful, his unspoken interest evidenced by his undivided attention, by his capacity for silence, by his facial expression, his smile, his gesture, and by his mere willingness to take the time to listen.

The cornerstone of psychotherapy lies in the *patient-physician relationship*. It is not amiss to recall that historically the physician or healer was once a priest. Sick people will turn to the physician with some of the old belief in his magical power to cure. Thus he is invested with attributes part of which are realistic, part unrealistic, that become powerful therapeutic tools. The patient brings to this relationship aspects of all the previous strong emotional relations he has had

with parents and others from childhood on. It is only to be expected, therefore, that together with his dependency and admiration, the patient may oft-times also feel resentment and fear.

In the treatment of these conditions the physician needs to know that hospitalization is rarely an adequate solution, that it may make the condition worse by providing too much attention, by putting a premium upon illness as a mode of reaction, by recourse to bed rest with its attendant increased preoccupations. These patients are best treated as ambulatory patients in repeated visits to the physician's office. Such visits can be brief; a half hour may suffice if the patient is not permitted merely to repeat symptoms but is obligated to search into causes. *The main tool will be language and conversation*, not medicine or laboratory procedures. The physician must recognize that he will play a vital and significant role, in which he will find the patient thrusts upon him or invests him with all kinds of emotional feelings, few of which are related directly to him, most of which represent the transfer to him of feelings, attitudes and emotional responses that had their origin long before in the patient's life. The physician must expect more than the positive emotions of respect and admiration. He must be equally prepared to accept the negative and hostile feelings with which the patient may be coping. He must not be thrown off the track by the patient's anger, resentment, criticism or dislike. Some such feelings may inevitably appear as a phase in treatment. Therapy begins with the very first contact with the patient, is maintained throughout the physical procedures, and hinges pre-eminently upon the process of history-taking.

Having met the immediate emergency, he proceeds with the *psychotherapeutic interview*. Numerous treatises have been written on the technic of the interview, all of which stress the importance of permitting the patient free, complete and spontaneous expression of his problems and needs. An interview is not the same as a social conversation. It is a process devised expressly to permit the patient to express anxieties without reservation to the physician, who will not interfere or hamper the spontaneity by injecting his own personality or his own convictions into the situation. In essence it is sensitive, objective, understanding, noninterfering, listening.

His first real task is to understand the origin and development of the anxiety in its chronology and in relation to the full activities of the patient's contemporary life, with particular seeking for evidences of dissatisfaction, conflict or anxiety relating to contemporary financial, occupational, marital, familial, social, religious and sexual tangles. Very soon he will see that the onset of the symptoms frequently coincides with periods of particular emotional stress or disturbing contemporary events in the patient's life. This is the present illness which consists of a step-by-step account of the appearance and development of the difficulty in its total personality setting.

Where the material spontaneously emerges, a rigid history review may not be necessary. The patient is told that for the physician's fullest understanding of the current difficulties, certain early biographic material must be studied. One begins then with the record of the patient's birth and the outstanding data of the infancy and childhood period, the school record, performance, and extent of education, the work record in terms of actual job successes, failures and reasons for change, the family status as to actual members and relations thereto, the place of the patient in the sibling group, and his emotional relationships with his family members. The marital history and status is obtained chronologically, with particular emphasis on the adjustment to husband or wife, the relations, or worries about children, the general satisfactoriness or unsatisfactoriness of the sexual adjustment. The general religious, racial and social adaptation as it relates to friends and community is surveyed for particular evidence of difficulties or failure of adjustment. The history of degree and extent of use of tobacco, alcohol, medication and drugs must be obtained.

Once these facts have been ascertained, and certain obvious areas of past or contemporary stresses have been revealed, what can the physician hope to do about them? Preeminently he may decide upon one of two procedures *to help the patient rid himself of his crippling anxiety, or to help him deal with it constructively, and perhaps to wall it off.* The physician may accomplish this in two principal ways, by helping to modify the unbearable situations of life, or by attempting to modify the patient so he can better tolerate the unbearable situation. Both procedures are included in what is called psychotherapy.

The physician may have found that the patient is faced with very real difficulties in his contemporary life. When these can be changed or alleviated, they should be. This may involve working with family members, to give them understanding of the illness, to relieve their anxiety about the sick person, to give them confidence in the therapist's attempt to cure the illness, to discuss the problem of the tangled emotional relations with husband, wife or parents, to help relatives to avoid overindulgence or overseverity, to act as marital counselor, to help with problems of emancipation, to act as impartial arbitrator in family disagreements or to counsel in the sexual problems of husband and wife to the end that they have mutual satisfaction.

Much can be done, too, in analysis and correction of unhappy, stressful work and school adjustments. Employers need interpretation and are avid for it, as is witnessed by the growth of industrial psychiatry. They usually welcome help in arranging work assignments and in understanding the workers' problems and needs. The physician can go far in the actual arranging of a better and more appropriate work or school placement. If he is in doubt of what to advise, he can get professional help through employment counselors.

Where economic and social privations play a leading role, the

physician can get help through the many social agencies in his community. More importantly, he needs to remember that every human being functions best within a framework of work, play and relaxation, which must be balanced. Attention to simple needs can bring much relief. The physician should insist upon regular exercise, vacations, rest, time for relaxation; the necessity of ending each day of work with mind closed to the task until the next morning. He may prescribe play; a movie, or social evening, or bowling, if need be. Activity is important; bed rest and unearned vacations accomplish little. Specific principles of relaxation can be taught the patient, such as vigorous exercise of a noncompetitive variety, prolonged warm tubs, showers, massage, or steam baths. The hygiene of sleep habits can be inculcated.

In brief, where obvious unbearable stresses exist in the actual contemporary life, they should be alleviated when feasible and practical. These technics might be broadly categorized as *environmental-manipulative*. Suggestions are in order, but it is well to avoid too much regulation. The wise course is to help the patient come to his own decisions and choices regarding major or critical changes in his way of life, and to adhere to this no matter how much the patient pleads for the physician's authoritative decisions.

Situational relief of the kind prescribed, while important, may be unfeasible, or may be at best a partially effective procedure. *Far more important is the effort to help the patient achieve inner security, freedom from anxiety, and an enhanced capacity to meet the actual stresses.* This is preeminently the talking therapy; psychotherapy in its more specific sense. The goal is to have the patient talk out, get verbal catharsis, release, get his "problems off his chest" with the aim of gaining greater understanding of his emotional life and difficulties. In this manner he may manage himself more wisely and free himself of his crippling anxiety. In the process, the physician will listen more than he will talk. It is not the physician's task to dig out confessions, or to discuss sexual experiences prematurely. His aim should be to create an atmosphere of trust and confidence wherein the patient will spontaneously bring into the discussion his sensitive, anxiety-laden experiences, memories and phantasies. It is surprising how quickly and spontaneously the contemporary difficulties give way to the discussion of the early life, with the heavy emphasis on the feelings and emotions experienced toward parents and brothers and sisters. With this recognition by the patient that he is transposing early feeling tones and traumatic emotions to contemporary people and situations, there may come a degree of insight that brings automatic relief from the present distress. More importantly, the healing comes automatically with the talking and sharing with the understanding physician. These are technics that might be designated as release therapy. Some degree of *explanation* to the patient of these mechanisms may be needed. Simi-

larly, explanations of the nature of his symptoms and how they are expressive of emotions may have to be given.

Hand in hand goes the need for *reassurance*. Here the physician's authoritativeness, and the patient's confidence gained through sharing, are of prime importance. Such reassurance may be necessary repeatedly in a frightened patient, but it should not be overdone lest it defeat itself by false and hearty optimism. It is more effective when offered sparingly and thoughtfully than too frequently and glibly. Properly used, it is a powerful and necessary tool, for it is the voice of authority and protectiveness. The physician should not promise too much, lest he lose face when the promised results do not occur.

This technic of reassurance may require added *persuasion*. In essence this means persuasion that the physician is right, that the patient must relinquish his problem to him, and that the symptom must be given up. This is particularly true if the secondary gain of illness is great, i.e., where the patient enjoys his illness, profits by it in over-attention, sympathy, or love, or finds it financially profitable in insurance or compensation. *Suggestion* must be recognized as a powerful weapon. Every physician commonly uses suggestion. He uses it when he shows confidence that his medication will be effective; when he completes his physical examination with assurance that his findings are negative. The wise use of suggestion can restore confidence and accomplish therapeutic relief. It should be emphasized, however, that suggestion is apt to be brief-lived in its effect and is only a temporary measure to carry over while more effective psychotherapy is being pursued.

The physician's increasing and full knowledge of his patient will inevitably sum up in his own mind as the need for *reeducation*. He will see how the personality is poorly balanced; how too much conscientiousness cripples performance; how excessive guilt feelings lead to insecurity and inadequacy; how foreboding anticipation cripples adventuresomeness; how habits of shyness, inarticulateness, distrust, exclusive self-preoccupation and lack of concern for others handicap social relations and friendships, how life-long attitudes of submission or aggression, temper or withdrawal, interfere with the fullest realization of normal health and behavior. Strength and courage and new direction will come to the patient mainly in proportion to what he can express and share with the physician. Haunting memories lose their stigma; fears and anxieties are relieved through desensitizing. The long distressing worry about masturbation disappears when it is understood as a developmental phase in every normal person's life. Similarly, other anxieties lose their charge through discussion.

The practitioner should inform himself of the excellent contributions made by the psychologist and the social worker, since both are usually available in his community and they are ready to add their skills to his study.

In brief, the physician who is not psychiatrically trained should limit

his technics to environmental manipulation, release of emotions, explanation, reassurance, persuasion, desensitizing and reeducation wherever these processes are utilizable at a conscious level. He should not try to uncover unconscious dynamic material, and he is not equipped to handle the explorative technics of narco-analysis, hypnosis, free association, or dream analysis.

Psychotherapy, therefore, can be employed at various levels by physicians of different training and backgrounds, and is a treatment procedure that can be employed effectively in the management of anxiety states, and indeed in the everyday practice of medicine by physicians.

PROGNOSIS

The prognosis for most anxiety states is good. With adequate psychotherapy, most of them can be expected to clear up. This is particularly true in the younger age group. With middle and late life patients arterio-sclerosis renders them more rigid and less modifiable, but even in elderly patients anxiety can be appreciably relieved by such technique. In general, these conditions have the greatest likelihood of recovery of any of the psychoneurotic reactions. They can be made irreparable worse by inept handling.

WHAT IS PSYCHOSOMATIC MEDICINE?

SYDNEY G MARGOLIN, M D * AND M RALPH KAUFMAN, M D †

PSYCHOSOMATIC medicine is an operational approach to the theory and practice of medicine in which the structure and function of the psychic apparatus are dealt with as a variable in health and disease, just as, for example, are physiology and pathology. It is apparent that this definition requires that the psychological factor be homogenized into every aspect of the approach of a physician to a patient and into the pathogenetic conception of the disease process. It specifically denies the type of thinking in which the psychological factor is considered as a separate layer or as an afterthought in the evaluation of the patient. To further implement this psychosomatic formulation of the theory and practice of medicine, an illness can be divided into the following phases with respect to the psychological factor:

- 1 The pre-morbid personality in terms of the social, economic, somatic and psychosexual adaptation of the patient, including the presence or absence of frank neurotic or psychotic symptoms
- 2 The onset of symptoms.
- 3 The latent period of the disease, i.e., the interval between the beginning of the disease process and the point at which the patient seeks help
- 4 The reaction of the patient to the therapist and to therapy
- 5 The reaction of the therapist to the patient.
- 6 The course of the disease
- 7 The post-morbid state, i.e., convalescence and recovery or invalidism

An essential point of these considerations is the elimination of the need to see the psychological factor as exclusively etiological or as a concomitant or as a consequence of an illness in any specific sense. The psychological factor is always present, but it may vary in its dominance and significance in the same disease, in the same individual and in every stage of the disease process. As a result, diagnosis and therapy takes on a rational character in that the timing and the nature of treatment and management becomes dynamically related to the interacting roles of the psychological, organic, and environmental factors at any given time. In other words, the psychological component

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may be weighted in relation to other factors, in such a way that it can become the major etiology, complication or sequel of the disease. This approach is in thorough-going agreement with the scientific thinking dominant in our current theory and practice of medicine, which is based on the concept of pathogenesis, i.e., the contributing roles of a number of different factors. As a result it is not necessary for the physician to revise his fundamental medical scientific thinking. Comprehension of the psychological factor, for example, should have the analogous influence that a concept of the role of the autonomic system has in the theory and practice of medicine.

As a result of a tendency to conceive of the psychological factor from an exclusively etiological point of view, it has become the fashion to consider such syndromes as asthma, gastroduodenal ulcer, ulcerative colitis and hypertension as the only representatives of psychosomatic illness. This, in effect, negates the fundamental thesis of the dynamic importance of the psychological factor in every aspect of a disease.

It is obvious, therefore, that psychosomatic medicine is not and never can be a specialty in medicine and does not require the designation of specific clinics, wards and departments in a general hospital. Of course, during a transitional period, pilot set-ups are justified for training and demonstration purposes. When the psychological factor has been integrated into the theory and practice of medicine, psychosomatic medicine as a term and as a unique point of view will cease to have any value. However, psychiatry and psychotherapy as medical disciplines will claim priority in the diagnostic and therapeutic management of the patient when the psychological factor is dominant in the disease, just as other specialties of medicine will be called in when the organic systems in their domain are the focus of diagnosis and therapy. Therefore, a psychiatric service will always be essential in the organization of a general hospital.

THEORY AND PRACTICE OF PSYCHOSOMATIC MEDICINE IN A GENERAL HOSPITAL

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Psychiatry for many years had been isolated in state hospitals and had almost completely lost its relationship to the field of medicine. As a result of the experiences of World War I, psychiatry became to some extent also an extramural specialty which, however, was still restricted to the diagnosis and treatment of mental disorders as such. Gradually, especially under the impetus of a psychoanalytic orientation, more attention began to be paid to the psychological aspects of illness. The interrelationship between psyche and soma, the meaning of illness, the patient as a person, the "comprehensive approach," and the other familiar platform planks of so-called psychosomatic medicine began to play an increasing role in the field. This resulted in the beginning of a rapprochement between psychiatry and medicine. There had been psychiatric clinics in the outpatient departments of general hospitals for many years. In addition the psychiatrist was used as a consultant by the other services. Some general hospitals set up psychiatric departments whose functions were poorly integrated into all the practices and services of the hospital. They served as minor psychopathic hospitals to which psychotic or disturbed patients were sent, usually out of considerations of manageability, rather than because of severity of illness. On the surface this seemed like an integration of psychiatry with medicine, but in actual practice there still existed an insulating wall which separated the psychiatrist from his other colleagues. In the outpatient departments this was not quite true, but nevertheless the bulk of the patients seen for diagnosis and treatment consisted of frank psychoneurotics.

Principles and Objectives.—The organization of the psychiatric service in a general hospital at any given time depends on the level of sophistication with respect to psychology. Therefore, no blueprint of an organization can be regarded as universally applicable. However, inasmuch as it is generally held that the psychosomatic point of view is not uniformly practiced, the following organization is designed for a general hospital with minimal existing psychiatric facilities. As will later be seen, its structure will be sufficiently dynamic and flexible as to permit revision, in terms of shifts of emphasis and foci of activity as the level of psychological indoctrination changes.

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The administrative set-up must be built around the professional needs of the institution. The *primary needs* are always:

1. Psychiatric services, i.e., diagnosis and treatment of the hospital population, both outpatient and inpatient.
2. Teaching, which involves two aspects—one, the further training of the psychiatric staff, and two, the indoctrination and teaching of every member of the hospital staff from administration through chiefs to house staff.
3. Research.

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These three functions should be regarded as separate chronological phases in the development of a psychiatric service in a general hospital. The attempt to install all three simultaneously at the beginning might result in an uneconomical expenditure of facilities and resources with the unfortunate consequence of failure. In support of this, our own experience has been that it took one year to establish the organization, a second year to develop a teaching program, and only in the third year have time and facilities made the initiation of a research program possible. It is when the operation of the organization becomes well established and relatively efficient that the personnel and resources become available for the second and third functions of the psychiatric service. Moreover, as will be seen later, the nature of the organization becomes a basic part of the teaching plan.

A psychiatric division in a general hospital should not be primarily for the treatment of clinical psychiatric entities such as schizophrenia, manic-depressive psychoses, and neurosis, but rather to be of service in every clinical department.

In accordance with these principles and objectives, the Psychiatric Service at the Mount Sinai Hospital was organized as follows:

Outpatient Service.—There is an outpatient service for adults which meets six mornings a week. This consists of three clinic units, each meeting twice a week. Owing to space limitations it is only possible to staff each clinic unit with a chief of clinic and eight psychiatrists. Hence, the three units have twenty-seven psychiatrists in all, each spending a minimum of six hours in two three-hour sessions. In addition a clinic meets two afternoons a week and functions as a combined follow-up and therapy clinic for patients who have been discharged from the inpatient psychiatric service. This is staffed by a chief and the residents. There is a psychologist and three social workers attached to the outpatient department.

The patients are seen by appointment, each appointment being for at least thirty minutes. The emphasis is on the quality of diagnostic and therapeutic effort rather than on the quantity of patients seen. The maximum number of patients to be treated by a psychiatrist during a three-hour period is five.

The patients are referred from the other clinics such as medicine

and surgery and from the ward services of the hospital. When accepted they are interviewed by the chief of clinic and assigned to one of the staff psychiatrists with consideration for his capacities and interests.

Child Guidance Clinic.—The field of child psychiatry and the emotional problems of children is a vast one. It is one of the most promising and needed subdivisions of psychiatry and yet, today, is the field that suffers from the greatest lack of trained personnel. At the Mount Sinai Hospital there is a Child Guidance Clinic which meets six times a week for a three-hour session. This is divided into two units with approximately six child psychiatrists attached to each unit, and the third unit is a special set-up for group psychotherapy with children and parents. There are three social workers, a clinical psychologist and a tutor on the staff of the Child Guidance Section. The patients for these clinics are referred from the pediatric service, the pediatric outpatient clinics and certain outside sources such as schools and family agencies. This section of the psychiatric service is the only one that has a direct contact with the community.

Inpatient Service.—The ward of the inpatient service consists of twenty-two beds divided into one large, comfortably furnished ten-bed ward for female patients, three two-bed wards and two three-bed wards. There is a recreation room and four treatment rooms. This ward service is staffed by two attending psychiatrists and two attending internists with special interest and training in dynamic psychiatry. There are five house staff members of resident status, one of whom is an assistant resident from the medical service who rotates for a period of two to three months. The service utilizes extensively the attending staff from all other departments as consultants. There are at this time six nurses, some of whom are graduates and some are students. Eventually there will be eleven. There are a full-time occupational therapist, a volunteer recreation worker, porters and attendants.

Each service in the hospital has a senior psychiatrist attached as member of its attending staff. He makes rounds with the house staff and, at specified times, with the attending staff.

Liaison Psychiatrists.—In the organizational phase of the psychiatric service under the minimal conditions that we postulate, it is probable that the activities of the liaison psychiatrists, of whom there are six in the Inpatient services, are of the greatest importance. In developing a healthy functioning rapport between the psychiatric service and the established traditional services of the hospital. Their functions usually begin with answering routine requests for consultations. However, the initiative of the liaison psychiatrist and the fact that he can contribute to the diagnosis and management of patients on the general wards soon makes him an invaluable part of the total therapeutic program. His efforts are consciously integrated into the problems of diagnosis and therapy. In numerous instances the psychiatrist cannot make a significant contribution. If this is so, he says so, and indicates why,

including ignorance as a reason. The essential attitude of the liaison psychiatrist in a general hospital should be one of collaboration and, above all, of not overselling.

These guiding principles operate with equal force in the activities of the liaison psychiatrist in the various outpatient clinics of our hospital. In a sense, the job of the outpatient liaison psychiatrist, of whom there are eight, is more difficult because of the larger number of patients concerned and the customary lack of homogeneity in the training and development of physicians in outpatient clinics. Moreover, it is in the ambulatory patient whose symptoms do not create an important incapacity but nevertheless a significant amount of discomfort and invalidism that the psychological factor frequently becomes the crucial issue. It is particularly in the outpatient department that the well known statement that "70 to 80 per cent of illnesses are predominately psychological" applies. Moreover, the liaison psychiatrist is in constant contact with the clinic physicians during the hours of the clinic so that his work is under continuous scrutiny. This, of course, increases the possibility of the exhibition of preformed prejudices on the part of the clinic physicians. As a result of these circumstances, the greatest test of the function of the liaison psychiatrist both as a doctor and as an individual often occurs in the outpatient department.

Admission of Patients.—No patients are admitted to the inpatient service directly from the outside. All admissions are screened and approved for transfer by the liaison psychiatrist attached to the particular service from which the patient comes. His basic consideration in approving such a transfer is concerned with the weight of the psychological factor in the diagnostic and therapeutic program for the patient. The medical and surgical regimen required for the patient is equally available on the psychiatric service as well as the specialized psychological management; the former through the attending internists attached to psychiatry and through consultants from the other services.

The Child Guidance Service which is intimately connected with the pediatric department has a group of liaison psychiatrists both in the hospital and in the outpatient department functioning along the lines indicated above.

Types of Cases.—The population of the psychiatric ward represents cases from all other services—medical, surgical and the specialties. Perhaps the best demonstration of the type of patient treated on the psychiatric service would be shown by a spot-check of the diagnostic categories represented at the time of the writing of this paper.

1. Ulcerative Colitis
Psychoneurosis (obsessive-compulsive with depressive features).
2. Ulcerative Colitis
Mental deficiency (obsessive-compulsive personality).
3. Manic-Depressive (depressed, hypochondriasis).

4. Essential Hypertension
Schizophrenia, catatonic.
5. Anorexia Nervosa.
6. Fugue State, questionable catatonic schizophrenia.
7. Ulcerative Colitis
Obsessive-compulsive personality.
8. Diabetes Mellitus
Psychosis with depression and paranoid trends.
9. Postgastrostomy
Hysterical personality type
10. Psychosis (paranoid state, somatic delusions).
11. Ulcerative Colitis
Schizophrenia, type unclassified.
12. Coccygodynia
Psychoneurosis, conversion hysteria.
13. Duodenal Ulcer—postvagotomy and gastroenterostomy
Passive-dependent personality.
14. Duodenal Ulcer
Passive-dependent personality in adolescent boy.
15. Rheumatoid Arthritis, Amyloidosis
Psychoneurosis, obsessive-compulsive.
16. Hyperthyroidism
Chronic anxiety state.
17. Gastric Ulcer—postvagotomy and gastroenterostomy
Psychoneurosis, hysteria.
18. Psychoneurosis (conversion, cardiac symptoms).

Teaching Potentials.—The educational program, both for the psychiatric staff and the hospital at large, is perhaps the most vital force that perpetuates the psychiatric service in the beginning phases of activity. The knowledge obtained through the psychiatric teaching is often the compensation both for the psychiatric and nonpsychiatric physicians. It is in the psychiatric service that the intraservice educational program is of the greatest importance, particularly because of the current diversity of schools of psychiatry and levels of training and achievement. Our basic orientation is psychoanalytic. However, because our theories and practices achieve the ultimate test in our contribution to the welfare of patients, we are able to achieve a common approach and common understanding in what appeared to be a diverse group of orientations. Again the emphasis is on the role of the psychiatrist in advancing the efficiency and functions of a general hospital.

Accordingly, with these objectives in mind, every unit of the psychiatric service has its own conference at least once a week. These are in the form of weekly clinic conferences, weekly liaison psychiatric conferences, daily ward conferences to replace the ward rounds, combined grand round conferences for all members of the psychiatric ward service, supervisory conferences between the attending and the resident and between the clinic chief and members of the clinic staff, conferences for supervision and discussion of problems of treatment and progress of individual patients. In addition, the training school for

nurses, nurses attached to the psychiatric service, social workers and other personnel are exposed to appropriate training programs in relation to the services they perform.

The second aspect of the educational phase is concerned with the physicians in the hospital as a whole. The brunt of this is carried by the liaison psychiatrists who, in the course of the exercise of their duties, are in constant contact with all the physicians of each of the services in the hospital. To this end a syllabus of a teaching program was worked out in a series of conferences by the liaison psychiatrists. Each instructor conducts seminars in which the following purposes are to be served:

1. Clarification of the meaning of psychosomatic medicine.
2. Discussion of the methodology of psychosomatic medicine.
3. Presentation of elements of psychoanalytic psychology including personality development.
4. Demonstration of principles of diagnosis and therapy from the psychosomatic point of view.

The didactic teaching is to be supplemented by demonstrations in the clinic, articles in the hospital publication and participation by psychiatrists in the staff meetings of other services. The liaison psychiatrists attached to the inpatient service conduct a seminar program for the house staff of the ward services. The objective behind the organization of the educational program is that every physician in the hospital should be given the opportunity of participating in the psychiatric training program.

Research Potentials.—With the incorporation of the psychological point of view in the functions of a general hospital, many problems will arise that lend themselves to different types of research, both clinical and experimental. As stated above, our own experience has been that only with the beginning of the third year were we able to initiate a research program which we hope will involve not only many members of the psychiatric staff but also members of other departments in collaborative projects.

This organization which applies to a general hospital with minimal psychiatric facilities and psychiatric indoctrination should adapt to its own success in introducing the psychosomatic point of view. For example, it is probable that the emphasis on the liaison psychiatrist as the spearhead of psychiatric indoctrination will shift to the clinic and research.

Summary.—A dynamic concept of what constitutes a psychosomatic program is presented. A report of a functioning psychiatric service in a general hospital is described.

ON THE NATURE OF STUTTERING

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THEORIES concerning the nature and etiology of stuttering continue to be presented in considerable variety. In 1948 Hahn¹ published a collection of papers by numerous students in the field. This volume provides an unfortunate reflection of the diversity of opinion on the subject. In the opinion of the writer there are two major factors responsible for the confusion. First, a not inconsiderable number of investigators have shown a disproportionate interest in the stutter at the expense of the stutterer. Second, quite a few of the papers apparently fail to take cognizance of or account for one of the most characteristic aspects of stuttering—namely, the classical variability of the appearance and the intensity of the symptom. It is well known, for example, that stuttering may become considerably alleviated if not abolished in some individuals if they are given a chance to act on the stage. In others the use of alcohol provides a similar effect. Singing in unison or speaking aloud to one's self may do the same thing. Conversely there are other situations—talking to superiors, denying accusations, speaking on the telephone, and so forth, which characteristically aggravate the symptom.

Theoretical explanations of the disorder should be able to account for the stutterer's capacity to speak flawlessly under certain conditions, yet this is often not the case. Orton and Travis, for example, have maintained that stuttering is due to a lack of cerebral dominance in one hemisphere. The forced shift of handedness from left to right is alleged to cause stuttering in some children who lack a dominant or master cortex. There are two serious objections to this contention, however. Investigations by the writer,² Despert³ and others revealed that only a small number of stutterers provided any evidence of a lack of a dominant hemisphere. Secondly, the theory fails to account for the stutterer's capacity to speak perfectly. Similar objections may be raised to other "mechanistic" explanations for the disorder—*asynkinesia* of symmetrical respiratory and laryngeal muscle groups, abnormal breathing, endocrine disturbances, allergic disorders, metabolic abnormality, disturbed electrical cortical activity, and so on. All of these explanations can be criticized on two grounds: first, that they again fail to account for stutter-free speech, second, no proof is offered that any of the abnormalities described are more than concomitant phenomena. It is noteworthy that control studies are lacking on non-stutterers exhibiting an equivalent amount of emotional tension to the stutterer.

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Heredity has been cited as a cause of stuttering. However, the writer who found stuttering occurring ten times as often in the families of stutterers as in the families of nonstutterers, was unable to identify the stuttering families with any clear-cut mendelian pattern. In the majority of families studied it was not possible to exclude such factors as identification, imitation or "influence" as responsible for the family incidence. At the same time it must be admitted that such a constitutional predisposition to stuttering may exist.

Physical and neurological examinations by the writer of over a hundred stutterers failed to reveal any significant difference between stutterers and normals, except for frequent manifestations of anxiety symptoms in the former consisting of dilatation of the pupils, sweating of the palms and soles, augmented deep tendon reflexes and sundry dermatologic-vasomotor phenomena.

These physical manifestations of underlying anxiety correspond to the findings of those students of stuttering who have approached the problem with a psychiatric orientation and who look upon stuttering as a varying disturbance of one of the major vehicles of expression as well as one of the chief tools in the establishment of interpersonal relationships. According to such an orientation it is as fruitless to regard stuttering as, so-to-speak, an *in vitro* phenomenon, as it would be to catalog smiles and scowls and grimaces without regard to underlying motivation.

Psychologic studies of the stutterer have consistently revealed that the stutter is but one outstanding and clearly perceptible manifestation of a *widespread underlying emotional disturbance*. Even a superficial anamnestic approach to the stutterer discloses abundant evidence of neurotic symptomatology. The histories of stutterers are replete with accounts of nail biting, bed wetting, emotional instability, anxiety feelings and catastrophic dreams.² Despert noted a frequent occurrence of intense fears, especially in regard to physical examination, in a group of fifty stuttering children investigated by her.³ She was similarly impressed by a high incidence of nail biting. Moreover, an investigation of the emotional climate in which the stutterer has been reared repeatedly reveals an emotional instability which is not conducive to healthy psychologic development. Despert noted that in the majority of her cases the mothers were domineering, overanxious and compulsive individuals while the fathers tended to be more passive and easy-going.

The mother of one of my patients was greatly disappointed at his birth, for she had hoped for a girl. The patient, her third son, was subsequently relegated to the care of a nurse. At the same time the mother resisted dressing him like a boy and allowed him to grow long curls. A highly neurotic, compulsive woman, whose major preoccupation appears to have been servant trouble, she became seriously depressed in later life and underwent several courses of electroshock therapy.

LACK OF ORAL SATISFACTION IN THE DEVELOPMENT OF STUTTERING

Detailed accounts of the dynamic mechanisms involved in stuttering have been made available through psychoanalytic studies. On the basis of such investigations stuttering can be shown to be the result of a conflict between a conscious wish to speak and an unconscious desire not to speak. The strength of the unconsciously motivated inhibitions of speech determines whether the stuttering will be mild or barely perceptible or severe enough to cause complete arrest of speech. The unconscious influences affecting the speech of the stutterer bear some resemblance to what takes place in a slip of the tongue. In the latter a consciously intended word is replaced by another which is unconsciously motivated. In stuttering an unconscious wish to suppress speech produces hesitancy or blocking. The unconscious motivation for the inhibition of speech may be due either to the specific content of the intended speech (e.g., the classical stutter of the marriage proposal) or to the symbolic meaning to the speaker of the general act of speaking. In the former case stuttering may be infrequent depending for its appearance on extraordinary situations. In the latter instance stuttering may exist as a chronic disability. In either case, however, stuttering is a function of interpersonal situations involving a disturbance of the act of communication between one person and another.

It is evident, moreover, that stutterers distinguish between that talking which is an expression of themselves and that which is not. For this reason many severe stutterers have had success on the stage, where, disguised and speaking borrowed lines, they may renounce responsibility for utterances which are but the vehicle for the expression of others. Yet behind the scenes they may be unable to ask for a drink of water without exhibiting their customary hesitancy. A similar phenomenon is noted in those stutterers who find their speech temporarily improved on emerging from a movie house in which they have identified themselves with some eloquent star.

A prerequisite for an understanding of the dynamics of stuttering is a knowledge of certain aspects of the psychophysiologic development of the infant. For some time after birth the relationship of the child to the world is in large measure a relationship between his mouth and a source of food supply whether it be breast or bottle. Repose and satisfaction are produced by successful and adequate nursing. Sucking brings contentment, while hunger, eruption of the teeth and other lack of mouth gratifications produce pain. Food and mother represent much the same thing and satisfactions from the one means satisfactions from the other. Adequate mothering or adequate feeding supplies a basic sense of security enabling the child to accept his physical separateness from the mother.

The process of weaning, whether from breast or bottle, can better

be tolerated by the child who has experienced consistent oral satisfactions. Assured of his ability to gain such satisfactions the emerging of a separate self and the unfolding of a wider universe do not present fearful prospects of isolation and abandonment but encourage the child to new achievements and increasing mastery. In time the mouth takes on a new function; through speech it serves as a means of communication, enabling the child to utilize this tool as a means of asserting his wishes and feelings.

A failure in the attainment of oral satisfactions brings pain, anxiety and hunger. Weaning and other measures which augment the sense of isolated separateness will be resisted and efforts will be made to gain what is lacking, even if it be at the expense of initiative and mastery. Thus there is engendered a tendency to prolonged dependence in which the mouth continues to play its original role of clinging to objects. Such dependence, however, is not without severe disadvantage to the individual, for it cannot be wholly accepted without a severe crippling of the child's ego, that is, his sense of self. Moreover, the environment will not lend itself, as a rule, to an undue dependence upon it. At the same time the development of increasing neuromuscular mastery encourages the utilization of aggressive gestures such as tantrums, screaming and biting to force from the frustrating mother-world the desired satisfactions. Yet aggression too has its limits for the child since it may alienate or even destroy the very objects it is designed to capture. Counteraggressive attitudes, threats, scolding or beatings may only further threaten the already shaky status of the child, providing further anxieties and a deeper sense of isolation.

Such alternating attitudes of increased helplessness and bursts of aggression become particularly evident whenever the child's security is threatened, notably at the birth of younger siblings. Such an event may precipitate a regressive infantile type of behavior and also hostile utterances toward the new rival. An early sense of precariousness or anxiety derived from inadequate oral satisfactions therefore promotes attitudes of mixed aggression and passivity which may permeate every aspect of interpersonal activity including speech. Under such circumstances words may take on an aggressive or sadistic meaning: they become weapons capable of perpetrating damage. Note the expressions "Words can kill" and "The pen is mightier than the sword."

Now the early history of the stutterer reveals with regularity just such a lack of oral satisfaction. Despert³ noted a high incidence of early feeding difficulties in the histories of her patients, as well as abundant evidence of significant emotional disturbances in the mothers. It is noteworthy, moreover, that gastrointestinal complaints are common among stutterers. One stutterer analyzed by the writer awoke nearly every morning with gastric distress. Another, a girl who had been a severe nail biter, weighed over 160 pounds at the age of eleven, which was clearly due to overeating.

SUBMISSIVE-AGGRESSIVE TRAITS IN THE STUTTERER

At the same time the stutterer provides ample evidence of prominent submissive-aggressive character traits.

One of my patients struggled constantly not to give in to secretly coveted helpless attitudes. He reacted with marked restlessness and anxiety, for example, when an ophthalmologist instilled homatropine in his eyes and made reading temporarily impossible. The helplessness of the situation forced him to finger the thickness of the waiting room magazines in order to guess the number of pages each contained. On arriving at his parents' home one summer he immediately contracted a sore throat, took to bed, and was catered to by his mother in a manner which would have been otherwise impossible. During an analytic session he "absent-mindedly" concealed the clock from my gaze, much as if to say, "Let this doctor-patient relationship endure timelessly." At the same time he recounted how he had torn the wings off flies as a child. In later years he had sexual phantasies involving a torture of the breasts of young women who were being sold at the auction block. Such phantasies were described by him with great reluctance. By and large his response to a sense of frustration by his wife consisted of some aggressive act ostensibly directed against himself. Thus when she asked him for money for clothes he responded that he would give it to her but added ruefully that he would be obliged to forego a suit for himself.

A similar pattern was disclosed by another patient who experienced violent anxiety in any situation which stimulated wishes to be cared for, sick or helpless. At the same time she possessed a "sharp" and "biting" tongue, assumed pseudomale attitudes and engaged in squabbles at the slightest provocation.

Rorschach studies by Krugman,⁴ performed on the same children examined by Despert, revealed consistent evidence of hostility and negativism. These and other investigators have agreed that the character structure of the stutterer closely resembles that associated with obsessive-compulsive neurosis, in which sado-masochistic elements are prominent. It is pertinent to note that Glauber⁵ observed favorable therapeutic results on young stutterers who while talking were permitted to "let out" their aggressive feelings by simultaneously sawing wood, cutting, or shooting off toy guns. It would appear that the Froeschels' "chewing method" of treating stuttering also serves to "draw off" oral sadistic elements of speech by allowing their expression through chewing movements. Undoubtedly some of the tics and mannerisms employed by the stutterer to help him initiate speech have a similar function.

The stutter therefore represents a defense, specifically a defense against forbidden aggression. The conflict to speak or not to speak runs parallel to the conflict between aggressive and submissive impulses, and in some instances to the conflict between sadistic and masochistic drives. It can readily be understood why stuttering is so variable a phenomenon in its appearance and intensity. Where speaking is "dangerous," i.e., before feared persons, and when aggressive impulses are mobilized, the symptom becomes aggravated. Before "kindly" persons, in moments of reduced sense of responsibility, for example, while

acting, or under the influence of alcohol, it may be lessened. To treat the disorder with drills and exercises seems futile for individuals who are already endowed with the capacity for perfect speech. Indeed there is some reason to doubt the wisdom of treating a symptom as an isolated entity when the symptom clearly plays an important defensive role in the total psychological structure of the individual. The development of two full-blown cases of schizophrenia in patients who were receiving largely symptomatic therapy for their stuttering² raises the question as to whether the threatened removal of a major defensive tool did not contribute to the outbreak of the psychosis. Interestingly, one of the patients was said to have lost his stutter in large measure after he was committed to a state hospital.

There is much more that is amiss with the individual stutterer than his stuttering. An effective and basic therapy ideally should be directed to the total psychologic problem.

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SCHIZOPHRENIA

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SCHIZOPHRENIA is a severe mental disorder, a psychosis, which is widespread in its occurrence. The largest proportion of patients in mental hospitals suffer from schizoplirenia. There are numerous other schizophrenic patients, impossible to calculate statistically, who are not confined to mental hospitals. Many of these patients have symptoms so mild, diffuse, subtle and indefinite that their condition is often mistaken for manic-depressive psychosis and especially for psychoneurosis. These atypical or diluted forms of schizophrenia will be discussed more fully in subsequent pages since they are the types often seen initially by the general practitioner. These cases should be recognized early before a great deal of time and money has been spent on misdirected therapy.

The terms "schizophrenia" and "dementia praecox" are often used interchangeably. Some investigators differentiate, however, between a deteriorating and a nondeteriorating form of schizophrenia. The markedly deteriorating or nuclear type they designate as dementia praecox, a process characterized by withdrawal from reality, incoherence of thought, loss of feeling, impairment of judgment, peculiarities of behavior, hallucinations and delusions, as originally described by Kraepelin of Heidelberg. The term schizophrenia they reserve for a broader concept than the term dementia praecox, to include cases in which deterioration is very slow or is completely lacking. The term schizoplirenia was first used by Bleuler of Zürich, who, even though in agreement with Kraepelin that many schizophrenics deteriorate, nevertheless widened the connotation to include cases which show a good tendency to remission or only a slow tendency to deterioration. He based the diagnosis mainly on fundamental observations made on the clinical psychology of these cases, stressing that both the nondeteriorating and the deteriorating cases show a disturbance of associations, formation of condensations, displacement of ideas, symbolizations, loss of ability for abstract thinking, a tendency to generalizations, impairment of the ability to reason, and affective alterations. Subsequently there may appear such manifestations as hallucinations, delusions and changes in personality.

Tredgold of England differentiates more sharply between these two types. The rapidly deteriorating group, he considers an organic disease, limiting the term dementia praecox to this group only, with an extremely guarded prognosis. For the more reactive group of patients,

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in whom the illness has been precipitated by some psychogenic conflict or stress and where psychological factors are most prominent, he reserves the term schizophrenia. Here the condition is not necessarily due to any organic change and consequently the prognosis is much better. For the most part, however, the terms "schizophrenia" and "dementia praecox" are used synonymously and interchangeably for the same condition.

The older clinical descriptions of schizophrenia emphasized mainly the deterioration, the presence of hallucinations and delusions, and the poverty or inappropriateness of emotions. More recent studies, however, indicate that there is a group of schizophrenic patients whose psychodynamic organization and emotional patterns are strikingly similar to those who show deterioration but who, nevertheless, remain in contact with reality for a long time. Although often behaving in a peculiar manner, they do not display such gross manifestations as hallucinations or delusions.

ETIOLOGY

The exact etiology of schizophrenia is unknown. Heredity and constitution play a role. Many psychiatrists feel that early environmental influences and psychological reactions of the individual to these influences play a large role in the etiology of schizophrenia but only in conjunction with an hereditoconstitutional predisposition. Specific neuropathological changes have not been found to explain the etiology of schizophrenia, although it is felt by many investigators that, in addition to psychological factors, brain changes will be found when more refined methods of investigation are discovered. At the present time there is a tremendous emphasis on the neurophysiological and metabolic changes of the individual brain cell.

About 75 per cent of cases of schizophrenia occur between the ages of 15 and 25. Schizophrenia occurs occasionally in children and some patients of 40 and over have manifested schizophrenic symptoms.

Although the exact cause of schizophrenia is not known, there are certain conditions of stress and strain which may precipitate a schizophrenic reaction in predisposed persons. Such conditions include toxic-infectious illness, severe exhaustion states, pregnancy and the puerperium, financial and domestic difficulties, unrequited love affairs, worry over sexual performance and sexual conduct, and the like. These conditions do not cause schizophrenia but may light up a schizophrenic process which is already present in a dormant state.

It is stated that schizophrenia develops in a specific type of personality called the "shut-in" or "schizoid" character. This individual is described as being seclusive, does not meet difficulties openly and frankly, has no friends, is apt to be a prude, with overscrupulousness and sentimental religiosity. He is apt to feel inferior, has no interest in sports or social affairs, has a tendency to drift with no particular drive

and is not interested in the opposite sex. He tends to be suspicious, jealous, sensitive to criticism and to blame his failures on others. There is reticence, stubbornness, lack of interest in outside things, and a tendency to live in a world of his own fancies and ideas. In addition to such personality type, the schizophrenic illness finds ready soil especially in the asthenic constitutional type. It may also occur in the athletic and dysplastic body type. Rarely does it occur in the pyknic type of constitution.

GENERAL SYMPTOMS

The general symptoms of schizophrenia, characterized by loss of feeling, withdrawal, and change of personality are so numerous and so diverse that it has been found helpful to divide them into four groups, namely, the simple, hebephrenic, catatonic and paranoid types of schizophrenia. The catatonic type may show either a catatonic stupor or a catatonic excitement.

- I. Simple Type.—1. Absence of any definite trend.
2. No hallucinations or delusions.
3. Lack of ambition, a general falling away of interest, chronic dissatisfaction, frequent change of jobs, tendency to idle shiftlessness.
4. Irritability, moodiness, asocial, preference to remain alone.
5. Memory well retained.
6. Chief feature is extreme apathy and emotional dulling.
- II. Hebephrenic Type.—1. Great incoherence in train of thought.
2. Delusions are fantastic and bizarre, auditory hallucinations are active.
3. Rapid shifts between excitement and depression or between different emotional expressions.
4. Silly, inappropriate laughter and smiling, often with peculiar motor activity.
5. Behavior is childish, silly, mischievous and playful.
6. Grimacing, posturing and stereotyped activities.
- III. Catatonic Type.—A. *Stupor*.—1. Sudden onset, mutism, marked degree of negativism or automatic obedience, refusal of food.
2. Position of universal flexion, sitting idly in one position with flexion of body at all joints, like fetus in utero.
3. Facial expression vacant, no interest in surroundings.
4. Requires complete nursing care, must be dressed and undressed, moved in bed, and tube fed or spoon fed for months on end.
5. Urine and feces often retained, requiring catheterization or enemas, or may be incontinent.
6. Saliva retained and putrefies in mouth or drooling occurs.
7. Insensitive to pain.
8. *Cerea flexibilitas* (waxy molding)—body and limbs placed in awkward positions are maintained indefinitely.

B. *Excitement*.—1. Sudden onset of frenzy of excitement, discordant activity, marked impulsiveness.

2. Stream of incoherent words.

3. Activity vicious, violent, reckless, assaultive.

4. May be homicidal or suicidal.

5. Active auditory hallucinations.

IV. *Paranoid Type*.—1. Commoner in women, occurs about age 35, occasionally after 40.

2. Personality retained for long period of time before deterioration sets in.

3. Ideas of reference, delusions of persecution, feelings of unjustifiable jealousy, ideas of grandeur, thought transference, special machines, active auditory hallucinations.

4. Feelings of being influenced from the outside by hypnotism, mental telepathy.

5. Feelings of being mistreated, food tampered with, poisoned or doped.

6. In accord with grandiose ideas, often decorate themselves very lavishly with all sorts of ornaments and insignia usually made by themselves.

7. Delusions are not usually fixed, but are changing and fantastic.

8. Deterioration is much less frequent and, if it does occur, is much slower than in other types of schizophrenia.

THE UNCLASSIFIED GROUP OF SCHIZOPHRENIAS

I. *The Pseudoneurotic Type*.—It was indicated earlier that there is a form of schizophrenia, without hallucinations or delusions, which is so widespread that it defies statistical evaluation. Often it is mistaken for a psychoneurotic condition and treated accordingly but with very indifferent results. Consequently we have designated this type of schizophrenia as the pseudoneurotic form. Other terms have been applied to this group, such as attenuated, diluted, latent, atypical, early, and mitigated schizophrenia. Many of the patients in this group do not deteriorate at all but nevertheless remain ill with a more or less nondeteriorating clinical symptomatology. There is no single symptom pathognomonic of this pseudoneurotic form of schizophrenia. There must be a constellation of related symptoms which together characterize this group. We shall briefly present the salient features. A more extensive discussion of this type of schizophrenia was published elsewhere.¹

Suspicious symptoms include a positive hereditary and constitutional history with an indication that from an early age there has been a marked sensitiveness to all environmental changes with the presence of free floating anxiety and panic reactions. More definite symptoms include complaints of not being able to reach people emotionally, of being unable to feel emotions, of being "dead inside," feelings of un-

reality, a constant feeling of fear and discontent, feelings of being dazed, and gross hysterical manifestations. These include fits, astasia-abasia, or vomiting without any reasons offered for these symptoms or without preceding tangible external reasons such as fright, trauma, compensation or criminal court proceedings as would occur in a neurosis. The presence of a panneurosis or pananxiety is characteristic for the pseudoneurotic form of schizophrenia. Here one observes psychoneurotic manifestations diffuse in structure and expressed in many different ways concurrently. This panneurosis or pananxiety will show hysterical features, neurasthenic symptoms, anxiety and phobic expressions and obsessive-compulsive phenomena. These symptoms may alternate but often they occur simultaneously. Frequently, the affect of these patients shows an expression of collective hatred with a tendency to be suspicious of every one. The patient's sexuality manifests a chaotic organization, with "polymorphous perverse" activities or phantasies.

II. *Acute Confusional States.*—This schizophrenic reaction manifests itself in a sudden onset with disorientation and confusion. The patient appears dazed and bewildered. His general attitude is dreamlike and the reaction resembles a delirium without the accompanying medical symptoms such as fever or other evidence of toxemia. This group also includes the so-called three-day schizophrenic reactions seen in the armed forces during the recent World War. Here there is sudden confusion with massive excitement or stupor, usually short lived. In these cases emotional precipitation is combined with situations of exhaustion. Hallucinations and delusions are usually present in conjunction with the delirioid reaction. A similar condition may occasionally be seen in postpartum or other exhaustion states and may be confused with delirious states of organic origin.

III. *Hysterical Reactions.*—There are schizophrenic states which resemble hysterical reactions and include such manifestations as amnesia, fugue states, hysterical fits and other hysterical characteristics, occurring however in the framework of schizophrenia. In these cases, the differential diagnosis can be made only after careful study.

IV. *Periodic or Cyclic Forms.*—Here we observe schizophrenic symptoms such as delusions, hallucinations, unreality feelings, peculiar behavior and emotional inappropriateness reappearing periodically, lasting a few weeks or a few months, and then disappearing, the patient returning to his premorbid level of adjustment and functioning quite normally. Because of the periodicity of this type it might be falsely diagnosed as a manic-depressive condition. This type is really a periodic form of catatonia.

V. *Schizo-Affective Group.*—In this group, the basic schizophrenic symptoms are mixed with manic or depressive features. Here the emotional shifts occur very quickly and this condition is often mistaken for a manic-depressive psychosis.

VI. **Depressive Forms.**—Here there is a large component of depression but occurring in the setting of a schizophrenia.

VII. **Symptomatic Forms.**—In this group, schizophrenic symptoms manifest themselves in the course of an organic psychosis, such as general paresis, alcoholic psychoses, hereditodegenerative diseases, and especially brain tumor.

PROGNOSIS

Schizophrenia is a serious condition and the prognosis, as a general rule, is guarded. In many, the course is downhill to ultimate deterioration. Some patients, however, make a complete recovery. Others show some evidence of deterioration and remain on this level for many years without deteriorating further. The pseudoneurotic form of schizophrenia may show no deterioration or its presence may be observed only after many years' duration of the illness. In schizophrenia, the following factors may indicate a good prognosis: acute and sudden onset of symptoms, short duration of illness, good prepsychotic personality, presence of physical and emotional precipitating factors, presence of a good affective response, clouding of consciousness, pyknic body build and a high intelligence. Age and sex of the schizophrenic patient cannot be relied on to give an accurate clue to the outcome. Of extremely poor prognosis are those patients with a history of gradual onset of illness over years without discernible cause in a schizoid or inadequate personality, who display little affect and show withdrawn behavior and illogical thinking in a clear setting.

TREATMENT

It must be kept clearly in mind that about 25 per cent of schizophrenic patients manifest a spontaneous remission. In spite of this fact it has been found that shock therapy provides a much higher rate of remission and consequently the most beneficial form of therapy in schizophrenia is one or another of the various "shock" methods. Insulin coma therapy is preferable in all chronic cases but is especially beneficial in the paranoid and hebephrenic types of schizophrenia. Electroconvulsive therapy (ECT) is more effective in resolving catatonic stupor and excitement, acute schizophrenic panics and the schizophrenic confusional states. Sometimes it is necessary to make the favorable effect more lasting by combining the electroconvulsive therapy with insulin coma. For best results, shock therapy must be given as early as possible in the course of the disease since the recovery rate is in inverse ratio to the duration of illness. Some schizophrenic patients will require, in addition to shock therapy, a course of psychotherapy. In others, psychotherapy can be omitted or can be quite superficial. In still other cases, psychotherapy is contraindicated, especially in the paranoid forms.

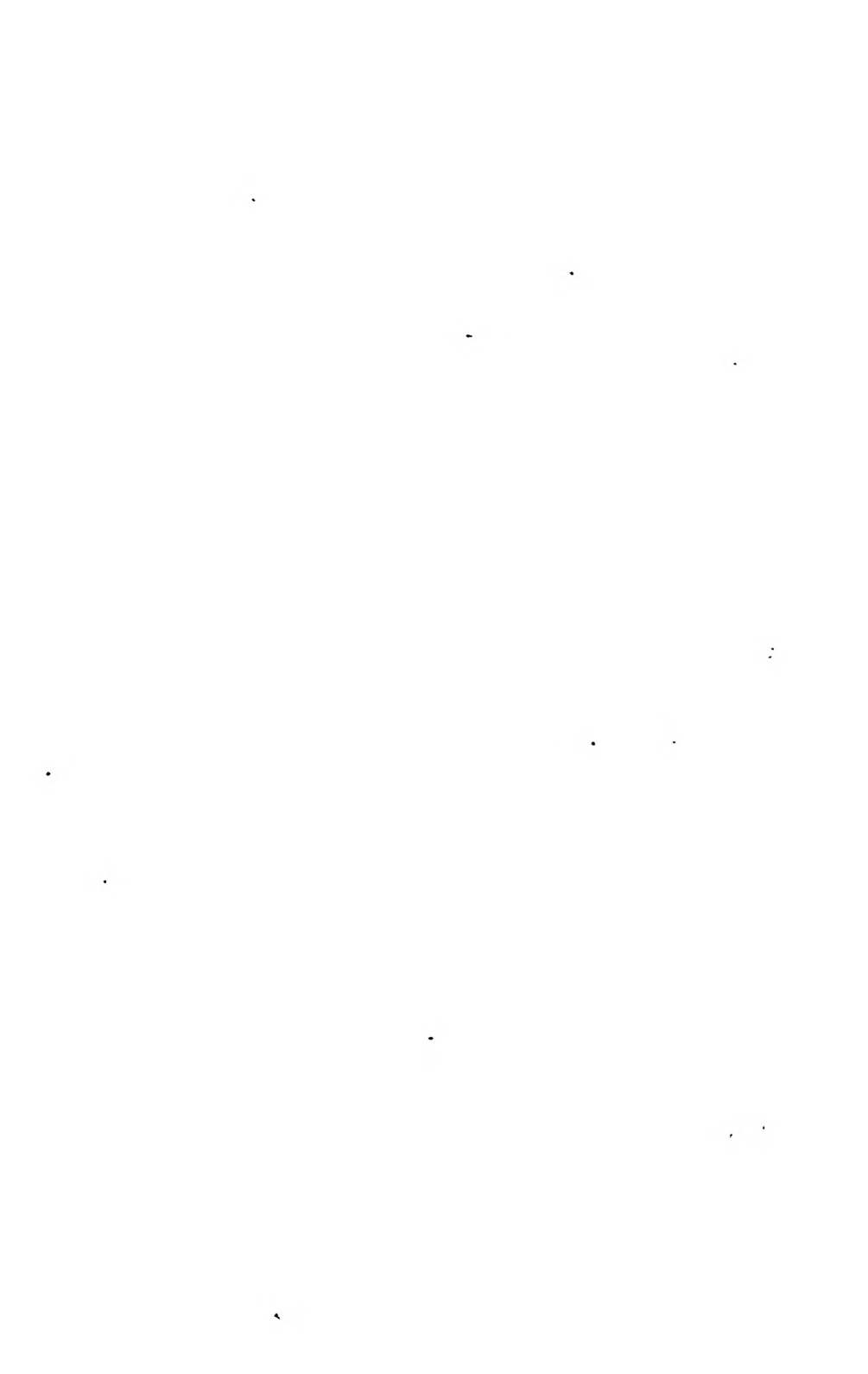
The pseudoneurotic form of schizophrenia, in many instances, is

very resistive to either shock treatment or psychotherapy. In some of these cases a prefrontal lobotomy offers promising results, but further investigations will be required before we can make unequivocal recommendations. Some very well preserved pseudoneurotic schizophrenic patients, however, respond well to intensive psychotherapy with a modified analytic orientation. In addition, they often require specific techniques like sodium amytal or sodium pentothal intravenously or ambulatory insulin.

Prefrontal lobotomy in chronic schizophrenia is being utilized but its effectiveness in such conditions is still debatable.

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BETTER DOCTOR-FAMILY COOPERATION AS AN AID TO THE MENTALLY ILL PATIENT

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To neglect the family is to neglect the patient, for many patients are inseparably linked to their families. There are ties of blood relationship, of living, of responsibilities, of mutual interest. The family is not only the source of the patient's genes and endowments, but its members have provided the atmosphere in which the patient was reared and trained. In the heat of the love relationships that existed and in the coldness of the internal struggles the pattern of his behavior was shaped, like steel formed and shaped by heat and hammering. They are the ones who may have recognized the symptoms of illness, and they may be responsible for referring and bringing the patient to a doctor. During the hours away from a physician or a hospital, it is the members of the family who provide companionship, who offer counsel, who inspire the will to get well, or who encourage dependency. The degree to which they inspire recovery or cultivate helplessness may determine the ultimate outcome of an illness. Thus it is that the physician in charge of any patient, particularly one who is mentally ill, must recognize and collaborate with the family.

To clarify the role of the family, the patient and the physician, let us consider the subject under three main headings: (1) how the family can assist the physician; (2) how they can best serve the patient; (3) how the physician can help the family. These three headings are inter-related.

HOW THE FAMILY CAN ASSIST THE PHYSICIAN

The members of a family are the source of the patient's inheritance; they have provided the surroundings in which he has grown up; they are the observers and the fellow actors in his drama of illness. Often they are intellectually and economically responsible for the patient's presence in the physician's office. It is, therefore, important in the initial interview, or at the earliest appropriate time, to talk with one or several responsible members. This affords the physician the opportunity of studying the people who are closest to the patient. One may note a meek, oversympathetic wife, who encourages her husband's neurotic traits, or one may encounter an aggressive, domineering type of woman, who keeps her husband in an insecure and inferior position.

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It is relatively easy to recognize an anxious mother, whose overprotective attitude is cultivating a neurosis in her child: she raises her boy in a timid manner; she instills anxiety and keeps him from making a recovery. Recall for a moment Elizabeth Barrett being detained as a paralytic by her father—"hopeless" invalidism from which she was finally rescued by Robert Browning. The patient's position in the family circle affords evidence to what extent the family can accelerate or retard recovery. Such observation may determine whether the patient should be treated at home or elsewhere.

Some psychoneurotic patients must be withdrawn from the home environment if they are to improve. Likewise, certain psychotics must be hospitalized, lest the neglect of a careless family endanger their lives.

The physician himself, if possible, or his representative (nurse, social worker), should obtain in this interview a history and a report of the family's observations. In some instances this can be brief, or may even be dispensed with, since there is adequate material upon which to arrive at a diagnosis and a prognosis. A long history would, then, be superfluous and inadvisable if time be precious. Very often, however, the data supplied by the family is a supplement to, a correction of, or more reliable than that supplied by the patient himself.

At times the family alone can give reliable and complete information. An individual who is referred because of a psychopathic personality may have as his major symptoms, pathological lying. The patient's history can be checked only through the statements supplied by others. Some patients forget their symptoms because of organic amnesia or a psychologic reluctance to recall an unpleasant event. Others conceal incidents because they seem degrading and shameful. Then there are those patients whose statements of grandeur or debasement may be truthful or delusional—whose stories of persecution could be fact or fancy. We depend upon the report of the family to appraise the value of these statements.

In the catatonic or negativistic patient we need an account of the subtle changes of behavior which finally led to the present stage of silence and withdrawal. A reliable history furnished by a relative can fill in the verbal vacuum left by the patient's silence.

No history of an epileptic is complete without the observations of those who have witnessed spells. The patient indeed may not be aware of his illness at all—the attacks occurred only in sleep; or the amnesia surrounding a seizure may cause almost total lack of appreciation of the illness.

It is also common for depressed patients to estimate their present symptoms as having persisted from earliest days. "I am dull, inefficient, a flop—I have always been a failure. I am the black sheep of the family—it's no use; no one can change me." Thus speaks the patient. Yet, when the physician interviews the family he hears, "She was always a

grand, capable person, she was dynamic and cheerful and ever so efficient. It's only in the last six weeks that she has become morose."

There are many other ways in which the family can aid in diagnosis. A patient may deny drinking or minimize his alcoholic intake. A husband or wife will correct this "oversight" of the partner by reporting the number of empty whisky bottles found weekly on the premises. I recall an instance of seeing a patient in deep coma in whom the diagnosis was in doubt. I was suspicious of drug intoxication. The family was asked to search carefully and reported that an empty vial of ten tablets of Veronal, 5 grains each, was found under the mattress.

HOW THE FAMILY CAN ASSIST THE PATIENT

The family can provide guidance, companionship and nursing care for the nervous and mentally ill patient. Only a certain number of psychiatric patients are resident in hospitals. I would estimate a greater number are at home during the entire course of the illness or in the pre and post hospital period. This applies particularly to milder cases and to those patients who are now receiving ambulatory shock therapy instead of being hospitalized for such treatment.

To improve the family's service to the patient, they should be instructed as to their specific duties in the matter of supporting the patient's ego, providing work and recreation, furnishing good food. Let us review these items in greater detail.

There is a tendency in families, especially on the part of parents with behavior problems in children, to expect and command the patient to conform to the performances of healthy members of the group. Again and again a person who is dynamic and successful will ridicule his faltering son with, "You've got it easier than I did—why can't you work? Why can't you be like me?" The robust, happy-go-lucky brother or sister of a depressed patient will say scornfully, "Cheer up—what have you got to worry about? Why can't you be like me, happy and smiling and singing even though I don't have much money?" Rarely does such exhortation have the desired effect. The patient whose eyes are filled with tears, who has a weight on his chest, whose legs are weak and trembly, hears the voices of his relatives and thinks, "Other people laugh. Why can't I? I feel so sad." He hears resounding words of cheer, but thinks, "They don't understand how sick I am. Guess it's no use—guess I'm a burden. I'd like to be like them, but it's impossible—no one understands." His depression deepens, his resentment rises. Listening would be better treatment than lecturing.

The family as well as the doctor must recognize the individuality of the patient, his traits and frailties, his weaknesses, even if they be emotional and not a fever or an amputated limb. Encouragement is always in order, but ridicule, scorn, and blanket enthusiastic commands—"Cheer up!" are not as helpful as they sound.

Instruct the family to be as objective as possible. This is much easier to write about than to attain. However, it may be possible for the family to learn to observe critically the reactions of a patient, just as they learn to take temperature, count pulse or test urine for sugar. The relatives of a patient who has some type of epilepsy may be taught to look for certain changes, such as muscle twitchings, that will help in the localization of the trigger zone. The family of a depressed patient can be helped to accept the illness more philosophically with such an explanation as, "If your mother had bronchitis, or tuberculosis, she would cough; such a cough is annoying to you, but you ignore it or endure it. You take for granted that a certain amount of coughing is an inevitable part of the illness. Your mother, who is now depressed, may sigh or groan or weep. Such symptoms are like reflexes, almost as uncontrollable in certain instances as coughing. Try not to let it get you down."

Most nervous patients, particularly those who are depressed, have a better sense of security if someone is at hand. The well member may make conversation, encourage card playing or needlework, may invite the patient to work, to go shopping, play golf, attend a movie. Frequently a depressed patient lacks initiative to get started, but once he is in motion, he may continue to make satisfactory progress. The companion may suggest or initiate some recreation. The patient should not be commanded, however, but coaxed or invited with such phrases as, "Come, let's take a walk to the corner," or, "How about the radio?" Invitations to a restaurant, a movie, for a drive, a visit, are in order. Comforted by the companionship of a friendly member of the family, the sick person may overcome his hesitancy, and once started, may be able to participate. The family can prompt and encourage activities which are beneficial in place of idleness charged with tension. They should realize that, "Action absorbs anxiety," and induce the patient to sew, clean, cook, garden, golf, and a hundred other recreational and constructive pursuits.

This applies also to the psychoneurotic. He needs encouragement and help to direct his thoughts toward constructive goals, away from introspective pursuits. However, derision and ridicule of so-called "imaginary" complaints are ineffective. They may prove a boomerang. It is far better for the family to listen to the symptoms, accept them, express confidence and hope, then to divert the patient's mind to a radio program, cards, conversation or some recreation or work.

If the mentally ill patient becomes dejected to the point of suicidal ideas, it is the duty of the family to provide safety until he can be hospitalized. If the patient be tense and disturbed by a sense of desperation, then he requires more than companionship. He must have constant vigilance. This is best provided in a proper hospital, but if hospital care is not feasible, it becomes the duty of the family to provide protection. There should be removed from his reach articles such as

sharp knives and firearms, which may invite self-destruction. Sleeping capsules and poisons, medicinal or otherwise, should not be available. The keys to the automobile should be taken away from the patient. Again and again near tragedies or hopeless catastrophes have resulted from failure to take precautions.

Mr. O. C. had been quite depressed and his family was worried about the need for surveillance. One day he disappeared from the house. The family found him in the closed garage about an hour later, slumped at the wheel of his automobile, unconscious, the motor running. He was resuscitated too late, for an irreparable damage to the brain had occurred.

Mrs. F. F. was examined at my office and found tense and agitated. Arrangements were made for hospitalization, but, unfortunately, two days elapsed until a bed could be obtained. The family was warned about the need for constant vigilance, and they informed me that they had removed everything that might be used for self-destruction—everything except a small bottle of lysol, which the patient had hidden in her pocketbook. She stepped into the bathroom, swallowed the lysol, and fell to the floor shrieking with agonizing burning in her throat.

Vigilance in the case of the depressed patient should not be relaxed even if he assures the family that he is now well.

Mrs. L. C. had been depressed for a period of several months. I saw her at her home in consultation with her family physician and the husband, and advised hospitalization with a view towards electrocoma therapy. Until the family was ready to take this important step, I recommended to the family physician that he prescribe a sedative to provide some ease for her insomnia. The doctor prescribed a large number (twenty-four) of 3 grain sodium amytal capsules. Two days later the patient announced to her husband that everything was better and she was all right again. (Constant vigilance had been impressed upon him.) The husband was overjoyed and took a long walk to the neighborhood stores. Upon his return he found his wife unconscious on the floor. She had taken the remaining twenty capsules. A gastric lavage was done and picrotoxin was injected intravenously. It was twenty-four hours before the patient was out of the shock and stupor of the amytal. Fortunately, a course of electrocoma therapy proved successful and brought about a complete recovery.

In another instance, a young man in the late teens who had been depressed for months, announced to his family that he was feeling fine. Everyone felt happy at this reported improvement. Some time later they were startled by a shriek and a fall. They rushed to the bathroom to find his lips and mouth brown from the contents of an iodine bottle. When the youth recovered, he offered this explanation: "For days and nights I battled with the idea that life was not worthwhile and I should die. During these days I was irritable and impatient. Early one morning I came to the conclusion that death was the only possible course, and I decided to end it all. This decision made me feel relaxed and comfortable, and I appeared so. Furthermore, I wanted to throw the family off the track. It was all an act to fool them, because I knew that in an hour I would have swallowed the iodine and would be asleep forever."

Such deeds are sometimes committed by patients discharged from hospitals as improved.

Although depressed patients demand the most watchful attention, other mentally ill need counsel, encouragement, and sometimes supervision. The youngster who is somewhat bewildered about life needs the

sympathetic ear of an older member of the family. The older, and particularly the senile, patient may lose track of time and place. Such patients quite commonly start for some destination, and then get lost. They may be unable to recall the street or number of their house. If this occurs, it is important for the family to accompany them on their walks. Recently a patient with paresis lost her way when returning home from work. Only after this occurred twice in succession did the family become alarmed.

Sometimes the supervision must be directed to the articles of food that the patient eats and to the matter of elimination. Patients with mental illness are likely to neglect eating because of the delusion that they do not have the organs with which to digest food, or perhaps with the delusion that the food is poisoned. As a rule, when such natural functions are seriously affected, the patient is best off in a hospital.

HOW THE DOCTOR CAN HELP THE FAMILY

The family which is confronted with mental illness is bewildered, frightened and troubled by many conflicting ideas. They cannot grasp the meaning of the illness; they are horrified at the thought of a mental hospital; some dread the word, psychiatrist. In some instances they feel a sense of guilt lest they caused the trouble, or they may fear that a similar illness will befall them or others in the family. Hence, to relieve this anxiety, the doctor should discuss the nature of the illness, its prognosis, and should provide such instruction as the specific situation requires. It need not be too specific, for a long period of observation may be required to settle the ultimate aspects. The need for a specialist may be pointed out, and he will be responsible for supplying a more extensive explanation.

Families rarely face mental illness with calmness or realism. Although they are aware that there is something wrong, there is a tendency to minimize the seriousness of the difficulty (wishful self-deception). "Yes, our son sits around the house; he doesn't read the paper or look for work. Yes, he mumbles to himself and seems to talk back to some mysterious person, but really, there's nothing much wrong with him otherwise. He knows what day it is, he can remember accurately the telephone number and the street of the house we lived in five years ago—and that's more than we can do. He will listen to the radio and he knows what's going on." The family then comfort themselves and try to present a picture of improvement by itemizing certain performances which are normal.

It becomes necessary for the physician to stress the serious meaning of certain statements and behavior, even though some performances are normal. It has been my custom to explain to the family that when a patient is delirious, he is so confused that he doesn't know who he is, where he is, and may not recognize family or friends; he is obviously disturbed. Surely his answers are odd and incoherent, and he literally

looks "out of his mind," yet with proper treatment he may be perfectly normal within a matter of days. Another patient, who is apparently neatly dressed and talks correctly about impersonal subjects, is much more sick than the delirious patient, because, aside from his intact mental functions, he is preoccupied by abnormal ideas. When he sits and listens for a voice which tells him to beware of his enemies, when he hides from these enemies or makes plans to get revenge, he is suffering from an illness which may last months or years and which may endanger his life and the lives of those around him.

Although it may be necessary to confront the family with objective evidence that indicates the serious nature of the illness, yet, in the main, the physician can give a hopeful outlook about recovery in many cases. Even schizophrenia, which in the past was called dementia praecox, and conveyed a hopeless outlook, may not prove to be as serious as was once thought. Be as considerate as the facts permit. Remember that even quinine is best prescribed in capsules.

A favorable response to treatment and improved conditions in mental hospitals have given to the public a more hopeful outlook on mental illness. Even in schizophrenia we know that many persons will recover from an initial attack and remain well permanently or temporarily, for months or years, before another phase of the illness sets in. Not all cases continue onwards to dementia. Some patients exhibit symptoms of schizophrenia which constitute an acute temporary reaction which tends to clear up spontaneously or with treatment. We have learned that shock treatment can bring about a more speedy recovery in many instances, even though there may be later relapses or recurrences of the illness. Such hopefulness in schizophrenia should not, however, encourage an overoptimistic prognosis. The illness is still serious. This applies particularly to the form of schizophrenia which begins in early and late adolescence, schizophrenia of a type where the individual has not made a full adjustment to life and has withdrawn from reality or found fantasy life, with wishful thinking, a substitute for the collisions and clashes of reality.

Schizophrenia which appears in later life, in a person who has attained maturity, has a more favorable prognosis. I have seen patients with symptoms, apparently of schizophrenia, which appeared rather abruptly in the fourth or fifth decade, who received courses of electroshock therapy and who made splendid recoveries. This applies to patients who have schizophrenic illness plus mood change (schizophrenia loaded with affect).

The prognosis of a depression is good; there are many spontaneous recoveries, as well as recoveries accelerated by shock treatment. A patient who is treated can, in several days or weeks, improve remarkably. With electrocoma therapy, properly administered, many patients may return to work in a matter of weeks. Most depressed patients return to normal well-being gradually or quite abruptly, some pass over

into a state of mild elation, while a few leap headlong into a phase of manic overactivity. Complete and regular cycles of manic-depressive phases are far less frequent than recurring episodes of depression, between which are longer periods of average health.

The prognosis in organic brain disease depends on the nature of the illness. Disease caused by arteriosclerotic brain change has an unfavorable outlook. However, though the general trend be downwards, there are stages of improvement which may last for weeks, months or even years. Other patients have improved periods and have normal thoughts on many subjects.

The psychiatrist will have to appraise the situation and arrange appropriate treatment. He must take into account not only the illness but the emotional, intellectual and economic status of the family. Thus, one patient should be referred to a private mental hospital; another requires state care, while a third is best treated at home.

In the event it becomes necessary to refer the patient to a mental hospital, select the best that is within the means of the family. Then reassure them as to the quality of modern treatment in such a place. It has been my custom to furnish the family with a mimeographed form such as the following.

INSTRUCTIONS TO THE FAMILY OF A PATIENT GOING TO A MENTAL HOSPITAL

An appointment has been made for a member of your family to enter _____ Hospital. Attached is a folder from the hospital giving you directions about visiting hours, etc. Please try to bring the patient to the hospital between the hours of _____ and _____, as these hours are most suitable for the admission of new patients.

At the time of the entrance of a member of your family to the hospital, you will be expected to sign certain forms. Rest assured that the hospital will take care of the patient in the best way possible and that the forms which you sign are guarantees of your cooperation.

The hospital has several divisions, from an isolation ward for very disturbed patients to an open ward which is as free as any country club. The patient is placed in that ward which is required by his condition, and will be moved if there is any change. Improved patients, as you see, are permitted to go outdoors, to go to occupational therapy with perfect freedom. Those who are very sick are confined so that they can be kept under more careful study and observation.

Visiting should be a source of comfort to you and reassurance to the patient. As a rule, it is advisable for you not to visit for at least a week, until the patient gets settled, unless you are instructed otherwise. These sick, nervous patients ask that you take them home. Should you carry out their wish, you have undone what you and the doctors have planned for quite a while. If you refuse, it makes you and the patient feel badly.

When you visit a member of your family in the sick wards, you may notice that other patients are nervous and perhaps even carrying on by gestures or speech. Such activity is not contagious. Do not fear that the ideas expressed by one patient will hurt or contaminate the thinking of another. No doubt, the member of your family was put in the hospital because of sickness in thinking or feeling and acting. The treatments which will be given, as a rule, will help a great deal towards his recovery. It is this hope which you must have in mind and not the fear that the hospital atmosphere is harmful.

Some patients will receive electrocoma therapy. These treatments are very beneficial for those patients who require them. However, the treatments produce a clouding of memory so that, for a period of time, some patients become very forgetful. Fortunately, when the treatments are completed, the memory returns in a matter of several weeks.

It is suggested that you come in to see the doctor who referred the patient to the hospital at intervals of once a week, or less often if the condition is improving satisfactorily, for a personal discussion of the progress.

INSTRUCTIONS TO FAMILIES OF PATIENTS RECEIVING AMBULATORY ELECTROCOMA THERAPY

A member of your family is to be given a course of treatment. This treatment is usually successful in bringing about improvement or recovery from the nervous symptoms. Your cooperation will prove helpful towards recovery. The following instructions may assist you in providing the best possible help.

1. **APPOINTMENTS** The patient is to report to the office on the mornings of Tuesday, Thursday and Saturday at 9 00, 9 30 or 10 00 A.M., as will be designated by the doctor.

2. **ATTENDANTS** A member of the family is to accompany the patient, stay until the treatment has been completed, and then escort the patient home. At the office, this member of the family is to act as bedside companion during the period of awakening from the deep-sleep state.

3. **CARE TO BE GIVEN** The patient may have a small breakfast, such as fruit juice and a cup of coffee, or a snack up to two hours before treatment on the morning of treatment. At the bedside, the patient is to be watched until he is fully awake. Then he may be permitted to dress and return home. Most patients are comfortable and need no special care afterwards. A few are troubled by headache, muscle soreness or drowsiness. They may rest as desired, take aspirin, 5 to 10 grains as needed. Those who feel well and are eager to be occupied, may help around the house, take walks and do as much of their accustomed work as they desire.

4. **MEMORY DEFECTS** Many patients develop a clouding of memory during the course of treatments. They even have no recollection of having received the treatment. This memory disturbance tends to clear of itself a few weeks after the treatments have been completed.

5. **NECESSARY SUPERVISION** Some patients who are nervous and very depressed are troubled by morbid and self-destructive thoughts. As a rule, a few treatments relieve such thoughts and the danger is lessened. Nevertheless, if, during the patient's illness, he is troubled by such unhappy thoughts, *it is imperative that he be closely observed*. Any articles that may invite unhappy action, such as drugs, should not be available.

6. **EXPLANATIONS AND ATTITUDE TOWARDS THE PATIENT** The patient who is ill enough to receive a course of treatment is usually in need of companionship and encouragement. He may be depressed, worrisome and fearful. You should not make light of his distress, but give as much hope as possible. Sometimes patients doubt the value of the treatments, or may feel temporarily improved. As a consequence, the patient may refuse to continue with his treatment program. It is important for you to insist by persuasion or strong urging that the patient continue his course of treatment if advised by the physician. Success depends upon a complete treatment program.

In general, the physician and the hospital should reassure the family by deed and word that the best possible care is being given the patient. They will usually reciprocate by doing their part gladly.

SPECIFIC ILLNESSES AND SITUATIONS

In the case of *epileptic patients*, the family should be taught to protect the patient against bodily injury. They may be assured that, as a rule, a patient will awaken from an attack spontaneously. They should keep a record of seizures and also do their part toward a satisfactory program of medication.

The *psychoneurotic patient* needs encouragement, not excessive sympathy. He should be kept occupied by therapeutic or actual work activity.

Depressed patients need companionship, gentle encouragement and sometimes vigilance. The patient should be referred to a psychiatrist for ambulatory or hospital shock therapy.

Manic patients are likely to get out of hand and create a serious disturbance which may wreck their position and their resources. Early hospitalization or at least ambulatory shock treatment should be instituted.

The delusions of a *schizophrenic* should be understood. Try to help rather than contest or refute! Arrange for early psychiatric care.

COMMENT

In all illness the physician must deal with the family of the patient. This applies emphatically to the mentally ill, for the family can add considerably to the history, can provide an environment for care—before, after, or in lieu of hospital treatment. An early interview will give them a greater sense of confidence and will aid the physician in understanding the background as well as the symptoms of the illness. An instructed family will cooperate helpfully in providing physical care and a better atmosphere for the patient. They will be better prepared to cope with problems of diet, sleep and suicidal danger. The cooperation of the family will often decide the feasibility of ambulatory electrocoma treatment and will contribute to its success. Give the family hope whenever possible, for this will encourage their zeal, provide comfort, inspire joint efforts. Such hope is usually justified by the good results so frequently obtained.

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MANIC-DEPRESSIVE PSYCHOSIS

PAUL H. HOCH, M D *

THE term "manic-depressive psychosis" was introduced by Kraepelin, who demonstrated that manic and depressive attacks frequently occur in the same person and form a single disease entity even though they appear to be quite dissimilar. Even though Kraepelin clearly formulated this concept in 1896, Kahlbaum had earlier described cases in which mania and melancholia occurred in the same patient, designating these different clinical manifestations as stages of the same disease under the coined term "cyclothymia."

Kraepelin drew attention to the fact that patients suffering from this type of disorder do not deteriorate, and showed conclusively that the same person can have quite a number of attacks during his lifetime without showing any dementia.

ETIOLOGY

The etiology of manic depressive psychosis is still unknown but hereditary predisposition is considered the most important factor. Kraepelin believed that about 60 to 80 per cent show hereditary tainting. Rosanoff in his study of twins demonstrated that about 70 per cent of the monozygotic and 16 per cent of the dizygotic twins were afflicted. The disorder occurs more frequently in women than in men, the ratio being 70 to 30.

Physical anomalies in patients suffering from this disorder are not more common than in the average population. Intellectually, they are of good standard. Physical changes complained of are frequently the result of the depression, for example pale skin or scanty menses. Kretschmer found that most patients suffering from manic-depressive psychosis show a pyknic type of body build and most of them show a cyclothymic temperament. They are extroverted, sociable, establish good human contact, but show mood alterations.

The psychoanalytic school of psychiatry following Freud explains a depression as follows: "The super-ego has taken possession of the entire sadism of the individual, rages against a helpless ego that acknowledges its guilt and submits to punishment (Oberndorf). The manic phase is interpreted as a "flight into reality," a running away from the conflicts by incessant activity.

When the disorder arises without any definite precipitating factor, then the depression is termed "constitutional" or endogenous. There

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depressive or manic reactions occur in many different mental diseases. Periods of elation or depression occur in organic psychoses, in schizophrenia, in persons with psychopathic personality, and fairly often in psychoneuroses. Therefore, not every patient who shows a clinical picture of a depression or mania automatically belongs in the manic-depressive reaction state. Differentiation is important from a prognostic and therapeutic point of view. It is obvious, for instance, that if the manic or depressive clinical syndrome appears in general paresis, the outlook and treatment would be different than if the patient is a true manic-depressive.

The differential diagnosis can be only touched upon here. The organic states are differentiated from manic-depressive psychosis by the presence of intellectual impairment, memory defects and disorientation which do not occur in manic-depressive psychosis. In the latter the intellect is unimpaired regardless of how many attacks the patient had. At times the patient is so manic or so depressed that his intellectual functioning is interfered with, but this is due purely to the altered emotional state, while in organic psychosis the intellect is directly affected.

The differentiation from schizophrenia is at times difficult. Usually, however, the previous personality make-up and the presence of hallucinations and delusions of a bizarre or paranoid nature indicate the presence of a schizophrenic process. If the patient should show, in addition, intellectual or emotional deterioration, we are quite sure that the patient is a schizophrenic. Periodic manic and depressive features are not uncommon in schizophrenia, and are often the source of a false diagnosis.

In some psychoneurotic patients we sometimes observe depressions which are usually reactive in a sense that they follow some personal loss. Usually the intimate knowledge of the structure of the neurosis and of psychodynamic factors involved enable us to differentiate them from a manic-depressive depression, even though at times the differentiation is quite difficult.

Finally, I would call attention to a group of patients who are believed to be suffering from psychoneuroses or, more often, are considered as suffering from all kinds of physical diseases. The careful examination of these patients, however, discloses that they are suffering from a so-called larval, or physical, form of a periodic depression. These patients periodically complain of fatigue, irritability, weakness and diminished sexual desire. Often gastrointestinal disturbances such as anorexia, constipation, and abdominal distress are present. Many other divergent physical manifestations also occur in these patients, mainly in the realm of the vegetative nervous functions.

These patients are often treated for all kinds of physical symptoms and are considered neurotic. Actually, they are suffering from periodic depression. The physical manifestations of the depression are much in

the foreground, but the feeling of depression, though obscure, can be elicited by a thorough psychiatric examination

TREATMENT

The treatment of manic depressive psychosis is an empirical one because the etiology of the disorder is not known. In addition to supportive measures which were used in former times exclusively, like occupational therapy and hydrotherapy, today three main treatment approaches are used: organic therapies consisting of shock treatment, drug therapies, and prefrontal lobotomy, psychotherapies consisting of different forms of supportive psychotherapy and deep psychotherapy in the form of psychoanalysis.

The supportive therapies are of importance and are used in all hospitals where these patients are treated. They consist of giving the patients an adequate amount of *recreation*, and work in the *occupational therapy* classes. The proper dosage of recreation and occupation in a depressed patient is of great importance. The layman usually thinks that a depressed person, if exposed to sufficient amount of recreation or sent into a new environment (vacation) or if pressed to do work, will lose his depression. Actually many of these patients do things against a great inner resistance and suffer intensely when asked to perform or even to concentrate on usually pleasurable activities. Therefore, such a patient should be distracted, but on the other hand, not pressed unduly to indulge in recreation or occupation. When the patient improves, gradually more pressure can be exerted.

Hydrotherapy is beneficial and continuous baths are used in many institutions for persons who show an agitated depression, more commonly, however, they are given to manic patients, in whom it has a good sedative effect.

Sedation with drugs is also an important supportive therapy for patients whose depression is not so marked that they have to be hospitalized. Depressed patients usually need hypnotics to insure sleep and to reduce the anxiety and tension which is present in most of them. Today barbiturates are usually used. One of the best tolerated and effective is sodium amytal given in doses of 0.06 to 0.12 gm (1 to 2 grains) about three times a day. In addition, these patients will need from 0.2 to 0.33 gmu (3 to 5 grains) of some barbiturate to insure sleep. If the patient is willing to take paraldehyde or chloral hydrate, both are very beneficial as sedatives. Manic patients usually need a much larger amount than depressives.

Today we do not use sedatives extensively in patients who are hospitalized because the shock treatment has eliminated the necessity. Even in the milder cases treated at home the method of choice today is to give shock treatment.

The choice of *shock treatment* is electric shock. These patients usually need four to twelve treatments. About 85 per cent of the pa

lients suffering from depression respond to the therapy with recovery. The treatment is given in the form of grand mal seizures (never petit mal, which is ineffective) and should be given two or three times a week.

In manic patients the response is also gratifying if the treatment is given in the following manner: for the first two days, two treatments every day; after that a treatment every other day. By spacing the treatments this way, it is possible to break through a manic attack satisfactorily.

A few patients do not respond to electric shock treatment and remain in a chronic depression or in a manic state. In these, *prefrontal lobotomy* can be performed, provided they have received two courses of electric shock, of fifteen treatments each, without any result and have been sick for several years.

Drug treatment has been tried extensively in manic-depressive psychosis. Of the many drugs used two have some value. Ephedrine and its derivatives, benzedrine and dexedrine, exert some influence on depressions, speeding the patient up and reducing the tension he is under. We usually give it in mild depressions in conjunction with sodium amytal. Only mild depressions respond to the drug and even then in many instances the response is not lasting.

Fluodyne, which is a hematoporphyrin preparation, is used sometimes either by injections or orally in depressed patients. The results, however, are meager. Some other drugs, such as nucleic acid (malonyl nitrate), were also experimented with. In our hands no definite results were obtained.

Insulin, which is effective in schizophrenia, is not useful in manic-depressive psychosis. Small doses of insulin, however, can be used to increase the patient's appetite and weight and to reduce anxiety and tension.

Psychotherapy has been used for a long time in manic-depressive psychosis. Most patients benefit by ventilating their difficulties and by receiving support in solving their conflicts. In an acute depression or manic episode, psychotherapy is difficult because the patient has only a limited ability to establish contact with the therapist. It is claimed that if psychotherapy, especially psychoanalysis, is used between attacks it is possible to eliminate the tendency of the patient to have a new manic or depressive episode. Statistically, however, these claims have never been substantiated in a large number of patients.

The shock treatment, too, can only cut short an attack but is unable to prevent the recurrence of a new manic-depressive episode. At present there is no reliable therapeutic tool at our disposal which will prevent the occurrence of manic or depressive attacks, but we are able today to deal successfully in most instances with the attack itself, cutting the episode short and relieving the patient from his distress.

THE DIAGNOSIS AND TREATMENT OF DELIRIOUS REACTIONS

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DELIRIUM is an important type of acute mental disturbance encountered by physicians engaged in all types of medical practice. It is due to nutritive or circulatory malsupport of the brain and is characterized by disordered comprehension of the environment, disturbances of consciousness, disorientation, fear and hallucinations.

From the standpoint of cause delirious reactions may be classified as toxic or organically determined. A third type of psychogenic delirium is less frequently seen and occurs chiefly in hysteria and epilepsy. The term toxic psychosis is used to designate those deliria caused by toxins due to disturbed metabolism or the toxic action of ingested substances such as alcohol or the bromides. The functional insufficiency of such organs as the heart, liver and kidneys is reflected in quantitative and qualitative changes in the blood circulating in the brain and delirious reactions occur in the setting of cardiac decompensation, hepatitis and uremia.¹ The deliria occurring with diabetes, thyroid dysfunction, pellagra, pernicious anemia and eclampsia are also of this type. In pneumonia, typhoid fever, influenza and meningitis the concurrent deliria are sometimes called infectious psychoses.

Examples of the organically determined deliria are those occurring in the setting of cerebral arteriosclerosis, senility, cerebral neoplasm and brain damage resulting from trauma.

In the psychogenic deliria, orientation and comprehension of environment are disturbed and hallucinations occur. However, careful examinations do not reveal significant physical disorders as the cause of the mental disturbances. The patient's history frequently shows evidence of previous episodes of hysteria or the occurrence of epileptic phenomena. In others there are evidences of a poorly integrated personality. A study of the content of the hallucinations usually reveals a prominent element of wish fulfillment. A particular group of psychogenic deliria occurring in patients who have had their eyes operated on and in whom toxic factors could be excluded, was studied by Preu.² The psychotherapy of these psychogenic deliria is essentially the same as that of the physically determined deliria with additional emphasis on the study and alleviation of personality problems which existed before the delirious episode.

Individual susceptibility to the development of a delirium varies.

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Persons who have shown evidence of poorly integrated personalities or a readiness in developing states of disturbed consciousness, experience deliria with only moderate elevations of temperature or relatively small amounts of sedative medication. Some of these individuals, because of long-standing problems in personality adjustment, have depended on sedatives to allay their anxiety and tension. Children often become delirious with a physical illness such as one of the exanthematous diseases. It is thought that this is due to the fact that the child's psychological functions are less mature and well integrated. Delirious reactions are frequent in later life.^{3,4} With ageing the homeostatic mechanisms are diminished in their ability to adjust to the additional stresses imposed by physical diseases and injuries. The physiological mechanisms maintaining the support of the brain are more easily disturbed and delirium results.

The content of a delirium has been found to be largely determined by the personality of the individual.⁵ However, certain drugs may produce quite characteristic clinical features. The early recognition of these features is important as it should lead to the elimination of the substance causing the delirium. Examples of such characteristic features are the frequency of hallucinations of small animals in delirium tremens. Also the occurrence of dark hallucinations in toxic reactions to morphine and the presence of colored hallucinations in bromide deliria.

The significance of personality factors is evident in those patients who have shown a tendency to suspiciousness or jealousy before they experience deliria. The delirium seems to release these personality features and during its course the patient displays prominent delusions of persecution. Unfortunately such delusions may not disappear with the subsidence of the delirium. Older patients with the increasing rigidity of personality that accompanies ageing may show such an unfavorable outcome. In others the delirium is seen to release latent tendencies to the development of schizophrenia or manic-depressive illness. Manifestations of these so-called functional psychoses color the picture of the delirium and persist after it subsides. Delirious reactions occur in the course of functional psychoses. The importance of their recognition and correct treatment has been emphasized by Jameison and Wall.⁶

The content and degree of behavior disturbance seen in a delirium do not always parallel the degree of toxic disturbance, the amount of fever or the quantity of drug the patient has received. Here in addition to the personality determined factors already discussed the physiological integrity of the central nervous system is important. Romano and Engel,⁷ by means of electroencephalographic studies of the disturbed cortical functions in delirious patients, have shown a correlation between the level of consciousness and the electrical activity of the brain.

The early and correct recognition of a delirium is important so that its treatment can be integrated with that of the concurrent physical disorders.

SYMPTOMS, COURSE AND PROGNOSIS

Early symptoms of delirium are restlessness and uneasiness with increased sensitiveness to noise and light. Sleep is disturbed and irritability with a tendency to emotional instability appears. The patient's restless sleep is troubled with frightening dreams. Such an onset may be fairly rapid following an operation, the development of a physical illness or the administration of a drug.

As the delirium progresses consciousness is clouded and difficulties in the comprehension of the environment appear. Orientation for time, place and the people in the surroundings is disturbed. Attention is poorly sustained and thought concentration is diminished. Although some patients are euphoric or elated the more usual emotional reaction is fear. This fear occurs particularly in relation to the misinterpretations of sensory perceptions (illusions) and hallucinations. These disturbances in perception are most frequent in the visual field and at first may be experienced only as the patient is falling asleep or awakening. They are parts of dreamlike experiences which the patient may recognize at first as imaginations or dreams. The content of the hallucinations is often frightening, the patient visualizing animal forms or situations threatening his safety. Auditory hallucinations also occur and the patient tells of hearing himself called insulting names. Hallucinations of touch are experienced by some, particularly in toxic reactions to cocaine. Some patients experiencing hallucinations of position and motion believe their beds are moving about or that they are on a ship. In "occupational deliria" the patient carries out actions characteristic of his usual work with the content of his hallucinations and delusions being appropriate to such activity.

Fluctuations in the level of awareness are very characteristic. The patient may be correctly oriented during the day only to have nocturnal recurrence of fears, restlessness and hallucinations. This characteristic nocturnal occurrence or accentuation of symptoms is important in the early detection of a delirium and in correctly differentiating it from other types of mental disturbance such as schizophrenia or an early manic excitement.

With or without treatment the delirium may subside in one of these earlier phases. However, with progression the patient becomes noisy, more fearful, overactive and uncooperative. Delusions occur which are usually in keeping with the hallucinations and are shaped in their content by the patient's personality problems and life experiences. Such false beliefs are usually not fixed or woven together into any system. As the patient's fear grows he becomes increasingly overactive and this further disturbs the support of his brain.

Physical symptoms and signs are indistinctness of speech, tremors and clonic or tonic movements and convulsions. The patient may pick at the bedclothes and be incontinent of urine. The gait and other coordinated acts are disturbed. Dehydration, malnutrition and vitamin deficiency arise from the patient's failure to eat and take fluids properly. The patient's temperature is usually elevated and laboratory studies may reveal leukocytosis, albuminuria and glycosuria. In uremia, diabetes or eclampsia disturbances in the chemical composition of the blood and urine characteristic of these disorders are found. Tests of blood and urine reveal the presence of bromides⁸ or other medications that can cause a delirium.

If the patient does not respond to treatment, coma followed by death may terminate the illness. However with active treatment and a satisfactory response the prognosis for recovery from the delirium is usually good. Definite improvement is usually noted within a week of treatment, the patient becoming clear and quiet during the daytime with perhaps persisting restlessness and mild apprehension at night. Gradually this subsides and in those who have been fairly well adjusted in their personality functions before the delirium, complete recovery occurs. A delirium is usually followed by a more or less complete amnesia for the period of the disturbances in consciousness and orientation. The average duration of a delirium is usually from a few days to two or three weeks. Older patients with cerebral arteriosclerosis or senile mental changes may recover from the delirium with memory disturbances and emotional liability persisting.

TREATMENT

Treatment in general is determined by an evaluation of etiological factors, by the degree of restlessness, disorientation and fear and by the character of the hallucinations.⁹ Physical and mental examinations as thorough as possible should form the basis for the therapeutic program. Hydrotherapeutic measures or other methods of securing rest and sleep for the patient should be instituted. Also there should be treatment to promote excretion of drugs and waste products through the skin, kidneys and bowels.

Some patients, particularly older ones with arteriosclerotic changes in the brain and kidneys, rather readily develop delirious reactions from the use of bromides for sedatives. The elimination of this medication from the body is effected by the administration of sodium chloride in 6 to 12 gm. doses daily, with maintenance of adequate fluid intake. During this procedure it is advisable to check frequently on the level of bromide in the blood, so that the chloride does not release the bromide from the tissues into the blood stream faster than the kidneys can eliminate it. Even after the administration of bromide has been discontinued such a condition will be apparent in a rising blood bromide level, indicating a reduction in chloride intake.

In general it is best to avoid the use of chemical sedation. The barbiturates in some patients appear to accentuate nocturnal confusion and restlessness. If chemical sedatives have been employed they should be discontinued immediately on the detection of the earliest evidence of delirium. Too often the administration of such a drug is increased as the restlessness increases, thereby adding to the delirium.

If a chemical sedative becomes necessary as overactivity develops, paraldehyde given in 10 to 20 cc. doses by mouth or nasal tube is the sedative of choice. For oral administration the paraldehyde should be offered in iced fruit juice so as to disguise its taste as much as possible. It is excreted readily and thus does not accumulate in the body to add toxic factors to the delirium. Chloral hydrate in 0.6 gm. doses may also be used. If such sedation is employed it is best if possible to confine its use to night time. It is to be hoped that other readily excreted sedatives that do not have the unpleasant taste and odor of paraldehyde will be developed. Such sedatives would contribute to ease in administration and the comfort of those who come in contact with the patient.

The rectal administration of medications and other rectal manipulations such as giving enemas and taking temperature should be avoided as they may add to the patient's fear or produce undesirable erotic stimulation. For marked excitement it may be necessary to give hypodermically morphine sulfate in doses of 0.015 to 0.020 gm. ($\frac{1}{4}$ to $\frac{1}{2}$ grain) combined with scopolamine 0.4 mg. ($\frac{1}{150}$ grain). In prolonged deliria with restlessness and excitement scopolamine in doses of 0.4 mg. ($\frac{1}{150}$ grain) three or four times a day is helpful.

To accomplish the important therapeutic aim of keeping the patient resting as much as possible, the use of continuous tub (prolonged) baths is the most desirable sedative measure. The water is circulated through the tub at a temperature of 97.5° F. In some patients temperatures of 95.5° to 96.5° F. produce more relaxation. The patient may remain in the tub for several hours if his reaction is favorable. Important contraindications are the occurrence of unmanageable degrees of excitement or circulatory embarrassment. Wet packs may be used, but they offer an even greater likelihood, particularly in patients with delirium tremens, of unfavorable circulatory reactions. Also, in some patients there is an accentuation of fear because of the feeling of being restrained. Mechanical restraint, such as tying the patient down to the bed or immobilizing his wrists and ankles, is always to be avoided. The confused, fearful patient does not understand such measures. He struggles to escape, thereby accentuating his fear and further disturbs the already embarrassed circulatory support of his brain. This additional exertion may lead to physical collapse.

Among the more important principles of treatment is the maintenance of adequate food and fluid intake. If the patient accepts food

by which he should receive a soft diet of adequate caloric and vitamin composition—a diet that is easily assimilated and its residue easily eliminated. Frequent small feedings are desirable. Milk is excellent for substitution between meals. If there is a history of preceding dietary deficiency, if the deficit is protracted or if the patient has over-indulged in alcohol the parenteral administration of vitamins is indicated.

In dehydrated patients the intravenous administration of 1000 to 2000 cc. of 5 to 10 per cent glucose solution with 20 to 25 units of insulin given subcutaneously is recommended.¹⁰ In addition, by the parenteral route 50 to 100 mg. of thiamine hydrochloride and 100 mg. of nicotinic acid are advised.

identified, serve to reassure him. When there are no physical contraindications to the patient's being out of bed and it does not increase his restlessness, fear or confusion it is better to interrupt prolonged recumbency. In some patients, remaining constantly in bed may not only contribute to the precipitation of a delirium but also lead to the development of circulatory stasis or bronchopneumonia. If it is necessary to keep the patient in bed, massage, frequent change of position, and some of the exercises developed for avoiding postoperative circulatory disturbances may be of value in preventing such complications.

The doctors and nurses should frequently reassure the patient and offer simple explanations regarding his fears, delusions and hallucinations. They should not argue with the patient concerning these symptoms or take them lightly with a joking attitude. Rather, the patient should be encouraged to treat such experiences as vivid dreams which he will later regard as unreal.

The content of a delirium may reveal deep and long-standing personality problems. It is unwise to attempt the psychotherapeutic discussion of these problems during the delirium, when the patient's comprehension, insight and judgment are impaired. After recovery from the delirium one re-evaluates the significance of these features and undertakes the psychotherapy indicated.

The frequency of deliria in general hospital patients together with the fact that a large number of them recover from the delirium in a relatively short time if the underlying physical disturbances can be ameliorated, indicates the advisability of making adequate provision for their care in such hospitals. In this way transfer to a psychiatric hospital is not necessary and the patient receives adequate care and treatment. With the consulting psychiatrist acting as adviser, single rooms can be made soundproof and their windows provided with safety screens. These rooms should be simply but tastefully furnished and decorated. The equipment for sedative baths should be conveniently located, if possible in an adjoining room. With such accommodations and adequate nursing care, delirious reactions can be properly treated.

SUMMARY

Delirium is a frequent type of acute mental disturbance characterized by disorientation, fear and hallucinations. It is caused by disturbances in the nutritive and circulatory support of the brain. In the treatment of a delirium the important features are the avoidance of restraint and chemical sedation with reassurance, safeguarding the patient from self-injury, the use of sedative hydrotherapy and the maintenance of adequate food and fluid intake.

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BEHAVIOR PROBLEMS OF CHILDREN

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CLASSIFICATION

Behavior disorders of children have been variously classified depending to a large extent upon the orientation of the psychiatrist formulating the classification. Some have viewed the disorders primarily from the etiologic standpoint. Others have stressed the symptomatology and established nosologies on this basis. For the most part, the different classifications are basically similar. Regardless of the orientation it is well to emphasize certain fundamentals which operate to produce deviations in behavior.

We should not conceive of behavior disorders as being due either to certain stresses in the external environment or to constitutional or hereditary factors. The disturbance is the result of a complex interaction between the total personality of a child and the external environment. It is to be noted that it does not represent a mere summation of personality plus environment but, as already indicated, a *complex interaction*; the resulting product (behavior disturbance) is therefore frequently unpredictable. We often find apparently similar environmental situations in superficially similar personalities producing different varieties of behavior reactions.

Brief outlines of several of the more commonly employed classifications will be presented and the classification utilized at the New York State Psychiatric Institute will be more fully described.

Pearson in the "Oxford Textbook of Psychiatry for Practitioners," and English and Pearson in their book "Common Neuroses of Children and Adults" have formulated the following outline of children's disorders:

- I. Disturbances of Psychological Function by Brain Injury or Disease
- II. Disturbances of Sociopsychological Adjustment Due to Innate or Acquired Physical Differences
 - A. Differences in brain development (mental deficiency or superiority)
 - B. Differences in physical development
- III. Disturbances of Sociopsychological Adjustment Due to Emotional Conflicts
 - A. Anxiety states
 - B. Acute diurnal anxiety attacks
 - C. Nocturnal anxiety attacks (insomnia, nightmares, night terrors, sleep walking, and talking)
 - D. Chronic anxiety states and phobias

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IV. Psychogenic Disturbances of Physiological Functions

A. Disorders of visceral functions

1. Disorders of the functions of the upper gastrointestinal tract (oral zone)
 - a. Emotional disturbances of alimentation
 - (1) Chronic anorexia, either general or for specific foods
 - (2) Dysphagia, either partial or complete
 - (3) Digestive disturbances—the gastric type of conversion hysteria
 - b. Reactivation or prolongation of infantile pleasure habits
 - (1) Finger sucking
 - (2) Nail biting
 - (3) Compulsion to eat too much food or articles with no food value (pica)
 - c. Speech disorders
 - (1) Inability to articulate clearly because of a traumatic speech neurosis
 - (2) Inability to articulate clearly because of stammering or stuttering or lisping
 - (3) Mutism (delay in learning to talk)
 - (4) Inability to talk understandably because of the use of neologisms
2. Disorders of the functions of the lower gastrointestinal tract (anal zone)
 - a. Constipation or soiling
3. Disorders of the functions of the urinary tract
 - a. Enuresis

B. Disorders of motor function

1. Partial or complete limitation of motor function
2. Hyperkinesia
3. Involuntary movements (tics, convulsive disorders)

V. Disturbances of Social Adaptation

A. Aggressive reactions—chronic aggressive states

1. Stealing
2. Delinquency
 - a. Occasional delinquency
 - b. Of feeble-minded children
 - c. Neurotic or due to fear of punishment
 - d. As psychotic symptom
 - e. Environmentally conditioned
 - f. Due to lack of love

B. Inhibitions of social behavior

1. Physiological, i.e., limitations of sensory or motor functions
2. Intellectual, i.e., psychic limitations of intellectual functions
 - a. Limitations of general intelligence
 - b. Limitations of specific intellectual abilities (reading and other educational difficulties)
3. Sexual perversions (voyeurism, exhibitionism, sadism, masturbation and homosexuality)
4. Psychoses
 - a. Reactive depressions
 - b. Schizophrenia

Rickman, utilizing psychoanalytic concepts and paralleling more or less the generally accepted classification for adults, formulated the following categories of children's disorders:

1. Anxiety states (anxiety neurosis) occurring at the earliest in infancy but commonest at the crises of the *vita sexualis*, i.e., puberty and late adolescence
2. Neurasthenia, earliest in infancy, commonest in adolescence

3. Conversion hysteria, earliest at the phallic stage, i.e., about three to six
4. Anxiety hysteria, earlier than conversion hysteria, most common after the latency period begins, i.e., about seven years
5. Obsessional, earliest in childhood, common during the latency period but less common at puberty
6. Paranoid reactions, after the attempted resolution of the conflicts about the parents
7. Manic-depressive reactions, after failure to resolve the conflicts about the parents
8. Schizophrenia, earliest in early infancy, commonest after puberty
9. Pathoneuroses, earliest very early in infancy, not common during the latency period

A very thorough and complete presentation of the behavior disorders was presented by *Kanner*, who employs to a large extent the psychological concepts of Adolph Meyer. He describes three main sections of behavior disorders with subdivisions as follows:

First Section: Personality Difficulties Forming Essential Features or Sequels of Physical Illness

1. Anergastic reaction forms
(Malformations of the brain, amaurotic familial idiocy, tuberous sclerosis, mongolism, brain tumor, brain abscess, meningitis, parainfections and post-vaccinal encephalitis, epidermic encephalitis, lead encephalopathy, juvenile paresis, cerebral trauma)
2. Dysergastic reaction forms
(Delirium, hallucinosis, stupor, coma)
3. Sydenham's chorea.
4. The endocrinopathies
(Thyroid gland, parathyroid glands, pituitary gland, pineal gland, suprarenal glands, thymus, sex glands)

Second Section: Personality Difficulties Expressing Themselves in the Form of Involuntary Part Dysfunctions

1. The central nervous system
(Headache, migraine)
2. The digestive system
(Psychogenic disorders of salivation globus hystericus, aerophagia, regurgitation and rumination, psychogenic vomiting, localized "neuropathic" disorders of the digestive tract, constipation and diarrhea, encopresis)
3. The circulatory system
4. The respiratory system
5. The urinary system
(Enuresis)
6. The muscular system
(Tics, nodding spasms)
7. The special senses
(Quantitative changes of perception, misinterpretation of actual perception, hallucinations)

Third Section: Personality Difficulties Expressing Themselves Clearly as Whole Dysfunctions of the Individual

1. Intellectual inadequacy
2. Emotional disorders
(Jealousy reactions, temper tantrums, fear reactions)

3. Thinking difficulties
(Daydreaming, lack of attention, lack of concentration)
4. The disorders of speech
(Mutism, late acquisition of speech, faulty articulation, disorders of phonation, stuttering, congenital word-blindness, congenital word-deafness, motor aphasia)
5. Habitual manipulations of the body
(Thumbsucking, nail biting, other manipulations)
6. Faulty feeding habits
(Faulty table manners, ravenous appetite, perverted appetite (pica), lack of appetite, gagging and vomiting)
7. Sleep disturbances
(Insufficient and restless sleep, nightmares and night terrors, sleep-walking, excessive sleepiness and drowsiness, narcolepsy and cataplexy, inversion of the natural order of sleeping and walking)
8. Antisocial trends
(Disrespect of authority, lying, stealing, destructiveness and cruelty, truancy from school, vagabondage)
9. Sexual difficulties
(Masturbation, heterosexual interests and activities, homosexual activities, fetishism, sodomy, the child as an object of sexual practices)
10. The attack disorders
(Convulsions, epilepsy, pyknolepsy, breathholding spells, fainting spells)
11. The minor psychoses
(Parergastic reaction forms, thymergastic reaction forms)
12. Children's suicides

The classification in use at the *New York State Psychiatric Institute* is considerably simpler than the preceding ones mentioned, but nevertheless encompasses essentially the same material. The disorders are divided into two main groups, depending upon basic etiology:

- A. Organic Disturbances, Resulting from Structural or Physiologic Alterations of the Brain (Secondary Behavior Disorders)
This group would include those listed in the first section of Kanner's classification (anergastic reaction forms, dysergastic reaction forms, Sydenham's chorea, and the endocrinopathics).
- B. "Functional" Disturbances, Resulting from Reaction to Environment
 1. Habit disorders—appearing in the preschool child
 2. Children with neurotic traits, e.g., acute and chronic anxiety states, phobias, compulsions, conversion phenomena, tics, psychosomatic symptoms, etc.
 3. Conduct disorders (delinquent reactions)—chronic aggressive behavior, antisocial behavior
 4. Psychoneuroses
 5. Psychoses

Mixtures of A and B may frequently occur.

ORGANIC DISTURBANCES, RESULTING FROM STRUCTURAL OR PHYSIOLOGIC ALTERATIONS OF THE BRAIN

The organic states will be considered first. There are three types of organically determined disorders which are commonly observed in children; namely those associated with mentally defective, postencephalitic and post-traumatic children. The term "intellectual inade-

quacy" is perhaps to be preferred to "mental deficiency," although social and emotional inadequacies are usually associated with the low intelligence quotient in children. The post-traumatic and postencephalitic children may have low I.Q.'s, either as a direct result of the infection or trauma or the result of other causative factors. In general, one may speak of the "organic type of behavior" which consists of impulsive, aggressive, destructive behavior, hyperkinesis (organic drivenness), and frequently emotional lability. Oftentimes there is evidence of impaired judgment, reasoning power, ability to memorize, and to develop and formulate concepts. These latter symptoms may be closely related to the intellectual level of development of the child and to the constitutional abilities of the individual to mature emotionally. The extent of the brain defects and pathologic changes may be closely related to the severity of the symptoms. In many of the organic problems superimposed neurotic features may develop; this is frequently the case in the intellectually inadequate children and is an important consideration in the therapeutic handling of the child.

Intellectual Inadequacy.—The categories of intellectual inadequacy include the *idiot* with an I.Q. range of 0 to 25, the *imbecile* with an I.Q. range of 25 to 50, and the *moron* with an I.Q. of between 50 and 80. The group of children with an I.Q. of 80 to 90 may be listed as borderline while the children with I.Q.'s of 90 to 110 represent the average. Most of the behavior problems in the intellectually inadequate which are dealt with in the clinic are those which appear in the moron group inasmuch as idiots and imbeciles are usually in institutions and also are not sufficiently aware of their surroundings to develop anti-social behavior.

The Moronic Child.—The moronic child will show a retarded rate of learning personal habits, including habits related to cleanliness, excretory functions, dressing and the like. Social responsibilities will be delayed so that the ability to play by himself or with other children comes later than normally. When the child enters school he cannot learn as readily as the other children. He has a poverty of ideas, lack of reasoning ability, and inability to grasp the significance of facts, and to learn from events and experience. Although these children are generally emotionally immature they nevertheless do have feelings and sensitivities and may often develop marked reactions as a result of their awareness of their deficiencies. Occasionally in these intellectually inadequate children there is a characteristic slowing up of their neuromotor skills and at times a retardation or impairment of general physical growth. Occasionally the bone age of such a child as determined by x-ray will correlate rather closely with the mental age of the child. As causative factors of low intelligence one may find a variety of conditions including cerebral aplasias and gross brain defects, hereditary conditions (e.g., phenylpyruvic oligophrenia), encephalitic or other inflammatory involvement.

irregular features in the organic disorders which differentiates it from the electroencephalographic abnormalities observed in the environmentally conditioned disturbances.

The Epileptic Child.—One is uncertain whether the various behavior problems observed in epileptic children are the result chiefly of the child's reaction to his illness or the result of inherited personality traits. The term "epileptoid" personality is often used to characterize certain personality features observed in some epileptic children. This epileptoid personality is similar in many respects to the personality noted in the organic behavior disturbances. In addition to the characteristics already described for the organic type of behavior, epileptics are said to be very willful, stubborn and highly egocentric. Successful treatment of the epileptic condition with anti-convulsant medication usually is associated with improved behavior in the child. Because of this it has been suggested that children without the clinical manifestations of epilepsy but who exhibit "convulsive patterns" in the electroencephalogram should be given anticonvulsant drugs such as dilantin, phenobarbital, tridione, or even glutamic acid. Of these dilantin has shown the most promise. Phenobarbital in combination with benzedrine has also been of occasional value.

FUNCTIONAL DISTURBANCES, RESULTING FROM REACTION TO ENVIRONMENT

The functional disorders, which have as their etiologic nucleus disturbances in the parent-child relationship, are characterized by an underlying "sea" of anxiety which is manifest in different ways. A thorough or even adequate evaluation of the psychodynamic and psychopathologic aspects of the various reaction types cannot be presented in this paper. Freud's and the subsequent observations of psychoanalysts have very clearly indicated that symptom-formation is a defense of the organism to anxiety. This anxiety is psychogenically determined and results from certain conflicts which are largely unconscious and preconscious. When anxiety appears in a child or in an adult, certain mental mechanisms are automatically called into play to control this anxiety; if it cannot be fully repressed then symptoms of one type or another occur depending to a large extent on the character of the mental mechanism employed to handle the anxiety. The character of the mental mechanism employed further depends upon the personality structure of the individual. Therefore, one individual may channelize anxiety into conversion phenomena, another into the formation of phobias, or compulsive phenomena, or psychosomatic symptoms affecting various organ structures. This concept of symptom-formation is of importance in the consideration of therapy.

Habit Disorders.—Habit disorders are those behavior problems which appear in the preschool child and revolve about the development and training of the child in the performance of the major biological

functions. They include disturbances in feeding, elimination and sleep. Anxieties, protest reactions and negativism may be manifest along the lines of these different biological activities. Feeding problems may include vomiting, refusal to take food, excessive chewing of food, or prolonged mouth-holding of food. Disturbances in elimination may include constipation, diarrhea, phobias centering around the bathroom, hiding of excreta, incontinence of stools and urinary incontinence (diurnal or nocturnal). Disturbances in sleep functions may consist of inability of the child to fall asleep readily, other forms of insomnia as frequent awakening during the night, nightmares and night terrors. Other disturbances observed in a preschool child may be faulty speech development due to poor training or excessive anxiety. Thumbsucking, nail biting, excessive chewing and sucking of various articles, excessive preoccupation with genitals and masturbation may also occur, and be manifestations of anxiety or chronic tension. It is to be noted that most of the habit disturbances in a preschool child may be classified as neurotic traits. Many of these gradually disappear as the child grows older and enters school, due in large part to an easing of tension in the parent-child relationship as the child becomes more socialized and develops other interests.

Neurotic Traits.—Children with neurotic traits may exhibit phobias of various kinds, such as fears of animals, of the dark, of strangers, of specific localities; and excessive timidity and shyness. Compulsive behavior is frequently manifest in the child over 4 and may consist of excessive neatness, preoccupation with cleanliness, compulsive tics, or certain ritualistic performances. Acute anxiety and panic states are occasionally encountered and sometimes may be transient in nature or give way to a less intense chronic anxiety. The tic manifestations in children may either be of an hysterical or compulsive nature. They frequently make their first appearance between the ages of 5 and 8 and most frequently involve the facial musculature, eyes, eyelids and shoulders. Most tics disappear about the age of puberty and may reappear at intervals during adolescence when the child is under increased tension. At times a severe generalized tic formation involving even abdominal musculature and often associated with vocal tics such as grunts, barkings and the like may occur. This condition is called "maladie des tics" and may continue for a long period of time, even beyond puberty. The psychogenic origin of tics in these cases is sometimes in doubt. Conversion hysteria in children has not been common in recent years, but when it appears it occurs chiefly in the older child. The various psychosomatic phenomena such as headaches, gastrointestinal disturbances, chest pains, the various allergies and skin reactions are as a rule more frequent in older children. All these evidences of neurotic formation which have been mentioned are the result of anxiety and indicate that the factors producing the anxiety and not the symptoms themselves must be treated.

Conduct Disorders.—The conduct disorders (delinquent reactions) generally include the chronic aggressive states with all sorts of misconduct, ranging from the mild forms to the more severe misdeeds. These children characteristically show aggressive behavior towards their total environment including the home, the school and the community. They lie, steal, fight, have vicious temper tantrums, are assaultive towards other children, are disobedient, and truant from school. They exhibit hyperkinetic behavior, are very restless, have a low attention span, cannot concentrate well, do poorly in school in spite of an adequate I.Q., and try to dominate situations where other children are involved. These children seem to have no conscience, and are generally not sorry for their misdeeds; their only concern is whether they will be apprehended or not. This is in contrast to the attitude of some of the children with organic types of behavior problems, who show concern for their actions.

Van Ophuijsen has conducted a study of a group of conduct disorders and he observes that they characteristically occur in disrupted homes where the parents reject the child. These children are frequently foster children who are sent from one foster home to another, each successive foster-parent finally becoming "fed-up" with the difficult behavior of the child. The aggressive behavior of the delinquent child may be differentiated from the aggressive behavior of the neurotic child since the latter generally directs his aggression towards a specific object such as parent, rather than directing it diffusely towards all areas of environment.

Psychoneuroses.—Well established psychoneuroses exhibiting the same symptomatology and structure as noted in the adult are not diagnosed in younger children since the various components of the personality have not become fully developed, but occasionally the diagnosis is made in the older child over 8 years of age.

Psychotic Reactions.—Psychotic reactions in children for the most part include the schizophrenic psychosis and a form of the depressive psychosis. The latter is quite rare in children and generally occurs only in the child approaching puberty.

Childhood Schizophrenia.—This is a controversial subject so far as etiology is concerned but the symptomatology has been fully described by various authors. In general the symptoms parallel those observed in the adult schizophrenias. Potter has developed the following criteria for the diagnosis of childhood schizophrenia:

1. A generalized retraction of interests from the environment.
2. Dercistic thinking, feeling and acting.
3. Disturbances of thought, manifested through blocking, symbolization, condensation, perseveration, incoherence and diminution, sometimes to the extent of mutism.
4. Defect in emotional rapport.

5. Diminution, rigidity and distortion of affect.
6. Alterations of behavior with either an increase of motility, leading to incessant activity, or a diminution of motility, leading to complete immobility of bizarre behavior with a tendency to perseveration or stereotypy.

Bender states that the schizophrenic child reveals pathologic changes at every level and in every field of integration within the functioning of the central nervous system, including the vegetative, motor, perceptive, intellectual, emotional and social functions. Thus the disturbance in the behavior pattern is reflected through all levels and fields of integration. A history frequently obtained in the schizophrenic child is that apparently the child was developing normally up to the ages of 3, 4 or 5 and in some instances older, and then gradually the child exhibited regressive behavior such as disturbances in speech, mutism, bizarre activities, stereotypies and repetitious acts. Some investigators believe that childhood schizophrenia is based on organic cerebral change while others contest this view. Discussion of the pros and cons will not be taken up at this time but there is considerable evidence to suggest that psychogenic factors play a significant part in the development of the disorder. A follow-up study of childhood schizophrenia by Lourie, Pacella and Piotrowski revealed many interesting findings including the observation that many of these former children, even at the ages of 20 or older, did not mature physically or endocrinologically. The prognosis for cure or substantial improvement is generally poor, although approximately one third of the children, particularly those who developed schizophrenia after the ages of 7 or 8, were able to "contain" their illness and make a tolerable adjustment. The later personality of these individuals is quite rigid; they are unable to cope with difficult situations in life but they can adapt quite well to conditions which require routine performance and activity.

Treatment of the Functional Disorders.—*Psychotherapy* is of value and should be employed in the functional disorders (although it has very limited use in the psychotic reactions). There are two general approaches towards the treatment of the child; a direct and indirect approach. Direct therapy refers to the total treatment plan employed by the psychiatrist in his personal relationship with the child. Indirect therapy involves the various attempts to modify the environment as far as possible where such modifications are deemed advisable.

In *direct therapy* it is necessary for the therapist to establish adequate rapport with the child and to discover his interests. The child should be made aware that the therapist understands that he has problems, and worries in connection with these problems. It is usually important to impress upon the patient that he is visiting the therapist in order to be helped in solving these difficulties. After varying periods

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It is to be hoped that with the present revived interest in the relation between psyche and soma, the promise of the skin lesion as a unique opportunity for objective study will not continue to be neglected; and that the future holds something beyond the regurgitation of old concepts and the present uncontrollable statements and non-objective speculations.

* * *

Anatomically and physiologically, the skin is both a protective barrier and an interagent, a liaison tissue or organ between the individual and his environment. The skin contains many and diverse end organs of the central nervous system, and is acted upon and subject to the traction of the emotion-expressing muscles of the body (e.g., many facial muscles, platysma, pilo-erectors). The skin contains the vascular and glandular structures which respond most sensitively to emotional changes (e.g., blushing, paling, sweating, secretion of sebum).

The skin acts as the receptor of sensations of pain, touch and temperature. In addition to this, the skin plays an important role in sexual function, both as a recipient of sensual stimulation and by its own sensual appeal to the visual, tactile and olfactory senses of others.

Psychopathologically, the skin may be used as an expression of hysterical exhibitionism, as seen in many cases of dermatitis factitia; or as a masochistic or self-punishing mechanism, as in instances of neurotic excoriations; or as a projection mechanism of paranoia, as seen in patients with acarophobia; and as an expression of displaced libidinal discharge as seen in pruritus ani, vulvae and scroti, and occasionally in other localized or generalized pruritus. These mechanisms may occur in hysterical, obsessional, or schizoid personalities and may be important components in numerous skin diseases.

The type of skin lesion produced depends upon the conjunction or interrelationship of many factors. Apparently, one often decisive factor is the "constitutional" make-up of the skin; for example whether it is so-called normal skin or skin which has a predisposition towards or susceptibility for blistering, lichenification, pigmentation, hyperkeratosis, eczematization, oiliness, and the like.

As a result of the unique embryologic, anatomic, physiologic and psychologic structure and functions of the skin, its changes are frequently due to the sum or synergism of several factors, rather than to one specific etiological cause. However, it often takes an alteration in only one of a chain of factors to precipitate and/or perpetuate an im-

of time a transference relationship may be established between the child and the psychiatrist in which the latter becomes a parent-surrogate. Frequently, by assuming a neutral and friendly attitude and at the same time by playing a relatively passive role the therapist can permit the child to act out during the interview his emotional relationships with his parents. If the child, for instance, has an attitude of defiance, stubbornness or hostility towards the parents these similar attitudes may eventually be displayed towards the physician. These attitudes on the part of the child may have been responsible for considerable tension and anxiety and it is the aim in psychological work with children to alleviate this anxiety by release therapy and/or interpretation of these attitudes in relationship to symptom formation. Children under 8 years of age cannot express their feeling tones very adequately by verbalizations, and the medium of play activity and the use of projective technics are employed in order to permit the child to express by symbolism his aggressive drives, fears, anxieties and emotions. Projective technics include the use of drawings, painting, clay modeling and woodwork. Further amplification of the therapeutic technic may be obtained from the book by Fred Allen entitled "Psychotherapy with Children," the monograph by Anna Freud on the psychoanalysis of children, and various chapters in the book "Modern Trends in Child Psychiatry," including those by Despert and Mahler.

The *indirect therapeutic approach* which attempts to modify the environment is generally conducted by the social worker, under the constant guidance of the psychiatrist, and aims to reorient and re-educate the parents, the guardians or the relatives who may be directly and indirectly responsible for the handling of the child. Sometimes it becomes necessary to modify the school or community surroundings such as advising the family to move out of a certain neighborhood or enter the child in a different school. However, case work with the parents is most important and is generally a long and tedious procedure. Frequent and relatively lengthy discussions are held with the parents; their attitudes, emotional reactions to various situations, personality characteristics, marital and interpersonal relationships with other members of the family are all discussed. It is emphasized to the parents that therapy with the child also involves adequate discussions with the parents. Attempts are made to reorient and reeducate the parents gradually and carefully in their relationships with the child. Generalizations are seldom used with parents and are ineffective when employed. It is best that they be reeducated in terms of specific situations that arise from time to time.

Although the parent-child relationship is the most important single environmental factor which has to do with development of the child's emotions, other forms of indirect therapy besides that of dealing with the parents are indicated. These include socialization therapy, and

group activities, enjoyable forms of educational activities such as the use of story and radio programs, stamp clubs and the like. A controlled educational program or the placement of the child in a certain type of training school sometimes is of great importance. In this connection there is a group of children with specific disabilities in speech, reading, writing and motor incoordinations who frequently develop neurotic reactions as a result of these disabilities. These children may be considerably aided by a proper educational program of training.

Pharmacological therapy is generally of minor value. Benzedrine is sometimes used in the very active child where it will diminish his hyperactivity, or in the very sluggish child where it may at times increase his general output of activity. Barbiturates or bromides may at times be used for sedation. I would caution against the use of shock therapy in the neurotic type of disorders; it appears to have only a very limited value in the psychoses of children and should be used with great caution.

The use of boarding schools, foster homes or institutions is, in general, indicated only when there is an attitude of rejection on the part of both parents and this attitude cannot be altered by a program of reeducation or reorientation, or when the parents are highly psychopathic or psychotic. However, a psychotic child should preferably be removed from the home when other siblings are present.

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balance which results in a dermatologic disease. The interplay of factors explains why widely different therapeutic measures from any one of many different approaches (e.g., local, external, endocrinologic, chemotherapeutic, climatic, dietary, psychotherapeutic treatment) can and do produce similar results in the same entity. Each strikes at one particular link of a pathologic chain which must apparently remain unbroken in order for clinical disease to originate or to continue.

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The following is a brief discussion of the parts played by psychogenic or emotional factors among the many mechanisms which participate in the predisposition, precipitation and perpetuation of dermatologic diseases and syndromes.

CONSTITUTION AND HEREDITY

From the psychologic point of view, individuals are constructed along varied personality patterns. Occasionally, the pattern may be quite simple and characteristic; more frequently, it is manifold and complex. It is the opinion of the present authors that in general the influence of mind and emotions on somatic disease is likely to be strongest and most clearly evident in certain particular constitutional types of personalities; e.g., schizoid personality.

From the dermatologic point of view, constitutional and hereditary factors are very substantial components in the determination of why the skin should be the "organ of inferiority"; and perhaps also in the determination of the type of lesion or dermatosis to ensue. These hereditary factors are strikingly noticeable in the abnormal susceptibilities to such changes as seborrhea, acne vulgaris, baldness, psoriasis, bullae (epidermolysis bullosa), keloids, xeroderma pigmentosum, atopic dermatoses.

NEUROLOGICAL FACTORS IN SKIN DISEASES

Some neurological entities are associated with characteristic skin changes, e.g., tuberous sclerosis associated with an oily skin and adenoma sebaceum; Parkinsonism associated with oily skin and with paresthesias and their ensuing excoriations; palsies with trophic skin changes, ulcers and pigmentary changes; and herpes zoster with its concomitant vesicle formation, epithelial degeneration and sometimes necrosis. These illustrative entities will serve to demonstrate the direct correlation between neurologic pathology and characteristic dermatologic lesions.

PSYCHOGENIC OR EMOTIONAL FACTORS IN SKIN DISEASES

Psychiatric Factors.—There are few, if any, *primary* dermatologic lesions in psychoses. The lesions that are seen are usually secondary. They are generally self-inflicted; for example, either excoriations or

alopecia due to pulling and rubbing. Some cases of dermatitis factitia may be considered due to psychotic states. However (according to experienced observers), in some psychoses certain types of skin changes do appear to be somewhat more common than the norm. Thus, some authorities contend that hypertrichosis and acneform eruptions are more common in schizophrenia.

Emotional Factors.—The emotions have both a direct and indirect effect on skin. Blushing in embarrassment, pallor in fear, sweating in anxiety, pilomotor responses (goose-flesh) in terror, and itching on suggestion or overexcitement or embarrassment—these are everyday examples of the transient, direct effects of emotions on the skin.

However, the emotional part of our lives also plays a much more subtle and extensive role in the production of disease through its influences on the central nervous system, which in turn affects the functions of various other systems of the body: the cardiovascular, the endocrine, the gastrointestinal and the reproductive. These changes in other systems may perhaps have a cyclic or indirect effect in altering the threshold of resistance and the reactions of the skin.

More often, emotions have both a direct and indirect effect on the skin, as seen, for instance, in rosacea, where the predisposed flush areas are almost invariably involved. Even the normal superficial blood vessels in these areas become readily dilated on emotional stimulation; and given certain intrinsic weakness or predispositions or added circulating poisons or infections, they may later become chronically dilated. In addition, in rosacea there is often a disturbance in gastric secretion, frequently with resulting achlorhydria or hypochlorhydria; and the vasomotor systems of the majority of these patients react excessively to alcohol, spices and hot foods. All of these factors may be superimposed on a personality which not infrequently solves life situations by alcoholic escape or by the use of drugs. The presenting picture of this complex constellation of mechanisms can then be a rosacea. As stated, many chronic dermatological entities are due to such interlinked complexities.

Thus in such syndromes as rosacea, one sees quite clearly how, to quote Nolan Lewis, emotions can be the "predisposing, precipitating or perpetuating" factors in the production of somatic disease. In a neurotic personality, where everyday life situations cannot be solved with relative equanimity, illness is frequently used as a cloak for personal inadequacies. We refer here to the constitutional type of personality that reacts to emotional stress with physiological disturbances. In the precipitation of illness, we refer to the imbalance caused by emotional crises which may perhaps in some unknown way make the patient more susceptible to infection, lower his threshold to allergens, or interfere with his regular physiologic and protective functions. The onset of illness may occur when the patient is under

or intermittent nervous tension or as a result of emotional stress. Here again bodily functions may be disturbed and in some way result in lowering general and specific resistance to living agents such as bacteria, fungi or virus, and to nonliving allergens. Thus, emotional factors may play all three roles of predisposing, precipitating and perpetuating illness.

PSYCHOGENIC DERMATOSES AND SYMPTOM COMPLEXES

According to present knowledge and experimental proof there are few, if any, dermatoses that are primarily or directly psychogenic. There are, however, many *secondary* lesions produced as a result of psychotic or emotional disturbances. This is understandable, since the skin is accessible to self-manipulation, self-infliction, self-punishment, self-love, self-aggrandizement, exhibitionism, and as the whipping boy in sexual conflict, guilt, and retaliative punishment. The skin is also the organ which displays the majority of the changes symbolic of aging, of loss of sexual attractiveness and desirability.

From the psychiatric point of view, the conditions of importance in the production of skin lesions range from mild, transitory neurotic manifestations to severe, paranoid psychoses.

Acarophobia is that condition in which patients believe that they are infested; and that living parasites—worms, germs or bugs—are crawling under their skins, producing various paresthesias. In our material psychiatric studies of these patients reveal a clear-cut paranoid picture. However, most of these patients are able to live within social bounds because their paranoia is shunted into the dermatologic symptomatology.

Neurotic excoriations are produced by patients who feel some irregularity on the surface of the skin and are compelled to remove the irregularity. The patient senses a feeling of relief when able to accomplish this or to remove hairs or produce bleeding. This process usually occurs at bedtime, or on arising or in other periods of idleness; the time spent on excoriating varies from a few minutes to many hours. From the psychodynamic point of view, this represents a milder type or borderline form of paranoia, presenting in many patients an obsessive mode of behavior. In some cases, the nature of the acts may be somewhat similar to that which is seen in the common "squeezing of pimples" or expression of blackheads, which can have masturbatory components.

Trichotillomania refers to the pulling out of hair, usually from the scalp, less often from the eyebrows, eyelashes and pubes. The psychiatric mechanism is similar to that of neurotic excoriations; and the sexual conflict and guilt appear to play a large role. (The junior author has seen a severe case of trichotillomania and neurotic excoriation occur during an acute phase of paranoid schizophrenia. On repeated oc-

casions, the trichotillomania and excoriations ceased as the psychotic stage improved; and then reappeared as the psychotic phase increased in intensity.)

Lesions resulting from compulsive movements can occur in any site. They are frequently seen on the buccal mucosa, lips and tongue, and are due to biting, chewing, sucking and erotic manipulation. Secondary maceration and erosion of the skin and mucous membrane due to thumbsucking are also occasionally seen. We have seen skin lesions from compulsive movements or tics on almost every part of the integument—from the toes to the crown.

Excoriations associated with *acne vulgaris* will often cease as the patient's acne improves and as the patient's social life broadens. In some cases, this type of preoccupation with the acne may be the starting point of neurotic excoriations and may even be the precipitating factor of a schizophrenic episode.

Dermatitis factitia is that dermatosis in which the patient produces traumatic lesions (artefacts) on the skin in order to obtain sympathy, attention or compensation, or to avoid responsibility. The patient knows what he is doing, although he invariably denies his own role in the production of the lesions. However, the patient is often unaware of the deeper motives behind his self-destructive behavior. In the histories studied, the patients were obvious failures, either socially, economically or sexually, after first having had a taste of success. These patients use their skin lesions as a justification for their failure in life. According to Gillespie, the motives discerned were dodging of responsibility, getting of attention (as a compensation for neglect or as a reaction of a hysterical personality), excitement of pity, attainment of revenge, expiation of guilt, and attainment of masochistic and exhibitionistic satisfaction.

Glossodynia refers to painful or burning sensation of the tongue. Very often these patients suffer from *cancerophobia* and fear that the sensations in their tongue are symptoms of cancer. For some unknown reason, this condition occurs most frequently in women and usually about the time of menopause. According to Daniel E. Ziskind and Ruth Moulton, psychiatric histories of these patients reveal sexual frustration and maladjustment prior to menopause, with an increase in sexual anxiety at the approach of menopause. To these patients menopause represents a crowning threat to their sense of self-esteem as women, and for which they believe "the only solution is death." (The junior author has some reason to believe that glossodynia occurs in these women because they are preoccupied with the fear of death, fear of cancer and, more specifically, fear of cancer of the mouth, the organ which sustains life through eating).

Cancerophobia is a disease which occurs most frequently in individuals at or past middle age, and is of course not allayed by present-day newspaper and radio propaganda. In many patients who have

come to us with cancerophobia, it was found that a relative, friend or close associate had recently died or become ill of cancer. In most cases thorough physical check-up and reassurance is sufficient to relieve the patient of this fear, at least temporarily. However, in rare cases, cancerophobia may be a symptom of a psychosis such as schizophrenia, paranoia or depression; or of a neurosis, particularly an obsessive neurosis or anxiety neurosis.

Syphilophobia is, for the most part, a transient state which follows extramarital or other "guilty" sexual relations. It is associated with feelings of guilt (often related to sexual matters and infidelities) and of punitive retaliation. Thorough physical examination and repeated serological examinations usually are sufficient to reassure these patients, at least for a while. But this condition also may, on rare occasions, be a symptom of a deeper psychosis or neurosis.

Hyperidrosis is the excessive secretion of sweat. In many such cases, it appears evident that emotional stress or neurological disturbances play a great part. The excessive sweating is usually of the localized type and frequently occurs where the sweat glands are most numerous and greatest, such as the palms and soles, or in regions with apocrine glands, e.g., axillae, groins, under the breasts and around the anus. Hyperidrosis in itself is a troublesome and embarrassing condition, but it also may promote complications, most notably fungous infection of the feet and of the inguinal and perianal regions. These complications were particularly evident during the war, when the combination of emotional stress, poor hygienic conditions, and the damp tropical climate produced many recalcitrant fungous infections.

DERMATOSES IN WHICH THE PRECIPITATING AND/OR PERPETUATING FACTORS MAY BE EMOTIONAL

The pruritic dermatoses in which emotional factors may sometimes play a significant role include principally atopic dermatitis (disseminated neurodermatitis) and chronic urticaria.

Atopic dermatitis (disseminated neurodermatitis) is a striking example of a condition in which the attacks or exacerbations may be attributed to not one, but a constellation of etiological factors. Apparently, in these patients, the equilibrium of protection may easily be thrown off balance by a disturbance in one factor of the constellation; and patients with this illness are often particularly vulnerable to emotional stress because of their sensitive personality structure. (It is the opinion of the junior author [S. Z.] that the personality structure of these patients is fairly characteristic and is evident prior to the outbreak of the dermatitis.) Added to the personality vulnerability, the constitutional factor (the intrinsic inferiority of the skin as an organ) and the hereditary factor (susceptibility to sensitization by certain specific allergens) must be considered. In approximately 50 per cent of these patients, there is either a family history of allergy or definite evi-

dence of clearly allergic manifestations (such as hay fever, chronic rhinitis, conjunctivitis, infantile eczema, asthma or urticaria).

Which is cart and which is horse, i.e., to just what extent is the personality of these individuals: (1) merely a concomitant part of the syndrome; or is (2) actually a causal factor; or (3) a result determined by the recurrent episodes of disfigurement, disability, sleeplessness and nerve-racking suffering? These fundamental questions are unfortunately still unexplored by proper investigative procedures and remain matters of speculation. There can be no doubt, however, that such devastating pruritic episodes must be very detrimental and crippling to the personality of the patient. Whether these individuals are constitutionally "thin-skinned" or become so as a result of repeated illness, or what part each of these possibilities plays, are problems still to be solved. However, certain phenomena are common in these patients.

First the allergic factor becomes increasingly polyvalent or diffuse as the patient grows up. As the patients go from infancy to childhood to adolescence and to adulthood, skin testing may produce a greater and greater number of wheal reactions; often there are skin test reactions to so many allergens that it is impossible to ascertain those with clinical significance. Also, in older patients, dietary elimination regimens are even less helpful than in younger groups.

Second, some of these patients fatigue unduly, even under the mildest of pressures; and some observers believe that many of these patients contrariwise "go without stopping," i.e., that they do not have a normal sense of fatigue and thus apparently squander their reserves of energy. With the fatigue or the incessant activity, there may be an exacerbation of the dermatitis.

Third, these patients very frequently do remarkably well with a change in environment (without the *deliberate* elimination of particular allergens or change of emotional stresses) be it hospitalization, change of residence, and in particular, change of climate.

Allergic, psychogenic, emotional, climatic (protection from heat, humidity, wind, etc.) and other factors, all may undoubtedly participate in the production of the often seemingly miraculously prompt improvements following change of locale. *The study of these factors and their relative importance is the imperative problem in this very common, and in many persons fearful and permanently incapacitating disease.*

"Idiopathic" pruritus may be generalized or localized (as in pruritus ani, vulvae and scroti). Most authorities agree that emotional factors may play a very salient role in the production of pruritus, particularly pruritus about the genital, anal, oral and aural areas. In some cases, the mechanism of pruritus is that of displaced libidinal discharge; in others, the pruritus and its ensuing scratching and rubbing may express the discharge of hostility, aggression, and guilt with self-punish-

ment The sexual and orgasmic character of the itch crisis, with its high tension, fierce activity, pain pleasure content, and climactic cessation with relaxation of tension, has long been recognized by dermatologists (Jacquet, Darier and many others) In the treatment of cases of pruritus which are principally due to nonpsychogenic causes, such as fungous infections, metabolic disturbances, blood dyscrasias, infestations, drug intoxication, or allergy to foods, drugs, wearing apparel and the like, it is well to remember that emotional stress can precipitate, intensify and prolong the sensation, even though not primarily causal

Lichen planus is a dermatosis in which the psychogenic factor is stressed in many textbooks and by many specialists However, the specific psychodynamics are as yet unknown, nor is there sufficient corroborative evidence as to what other factors may come into play We have seen several cases of lichen planus in which there was evidence suggesting the perpetuation or exacerbation of the disease during periods of emotional stress Just as in psoriasis (see below), the inference is permitted that in some cases the psychic and emotional influences play the role of trigger mechanisms in bringing on the characteristic eruption (isomorphic response of Koebner?) However, the disease lichen planus appears to be due to an unknown infectious agent, and the course and exacerbations are often without apparent or direct relationship to psychic or emotional influences

Psoriasis is still one of the challenging problems of dermatology, both from the etiologic and therapeutic points of view Foci of infection, endocrine disturbances, liver dysfunctions, vitamin deficiencies, specific infections of the skin, as well as psychogenic disturbances, are among the many mechanisms which have all been advanced as causes of psoriasis There may be some truth in any or all these theories, but all are without substantiation From the psychiatric point of view, there are two indisputable facts First, in a small number of cases, the initial outbreak of psoriasis indubitably followed closely on some traumatic emotional experiences Second, in quite a large number of cases, exacerbations of psoriasis are chronologically related to emotional stress Thus the inference may be permitted that psychogenic and emotional situations can, in some instances, play a role as trigger factors in bringing out or localizing the psoriatic eruption, just as there are many instances in which known intercurrent infections, physical or chemical trauma and allergic skin reactions are seen to elicit or localize the psoriatic cutaneous response (isomorphic reactions of Koebner as in lichen planus)

Seborrhea is an inflammatory process, probably associated with hyperactivity of the sebaceous glands and favoring the scalp, the "flush areas" of the face, the chest and back, but often involving other skin areas It has long been held by dermatologists that the sebaceous glands are under nervous and central control and in addition are sub-

ject to endocrinologic influences. It is also believed that avitaminosis, particularly of certain factors of the vitamin B complex or of vitamin A, is sometimes an accompaniment of seborrhea. As the cutaneous vasomotor reactions and the secretory responses of the skin glands are under nervous and emotional control, this undoubtedly may play some part in the symptom-complex "seborrhea," just as it may in acne and rosacea (see below).

The oily facies ("salve" or "ointment face") in post-parkinsonian states, is a striking item of evidence in this regard. However, there is unfortunately no exact information on the mechanisms of these relationships, and no practicable therapeutic or preventive approach via the endocrinologic or psychic spheres.

Rosacea is an inflammatory process which usually affects the flush areas of the mid-face, and sometimes the chest and other parts. Many individuals afflicted with rosacea have labile neurovascular systems, characterized by facile vasodilation and easy flushing and blushing. It is therefore understandable that the rosacea is often aggravated by extraneous vasodilators, such as rapid changes of environmental temperatures, alcoholic beverages, hot food and drink, coffee, tea, spices; and also by emotional and psychogenic effects upon the neurovascular and glandular systems of the skin. Achlorhydria or hyperchlorhydria are such common accompaniments of rosacea that this suggests that there may be some relation between the gastric and cutaneous reactions.

Acne vulgaris is almost (or perhaps entirely) physiologic; and only the exaggerations in degree, extent, location or duration constitute a true disease. Acne usually makes its appearance about the time of puberty, when the individual undergoes the most dynamic changes from childhood to adulthood, both physically and psychologically. The general body growth and development reach their peak of activity. The reproductive system begins to develop rapidly from its latent infantile state to the mature sexual level, with accompanying widespread secondary changes through the influences of the hormones. One of these changes is the radical alteration of the skin of the child to that of the adult. This change includes the tremendous development of the pilosebaceous apparatus and growth of the skin's hairs, glands and horny covering. In patients who have a tendency to develop acne, this transition period is characterized by enlargement and overactivity of many of the oil glands and by plugging of their orifices and dilatation of their ducts. Apparently these structures then also become more vulnerable to infection and to irritation by certain chemicals, particularly iodides, bromides, oils and chlorinated diphenyls.

Accompanying these physical and functional somatic changes, there are important psychological changes. With sexual development and awareness come sexual needs and desires, with their ensuing problems. Added to those are the social and educational pressures. The child has

to give up childish play and meet maturer competitive life situations. These new problems are not made easier by the frequent outbreaks of acne lesions around the time of school examinations or an important "date" or "party."

Because most cases of acne occur during the crucial age of adolescence in which poise and physical appearance are extremely important to the individual, and because most of the disfiguring lesions appear on precisely those sites which cannot be covered and hidden, the acne itself has a tremendous effect on the personality of these young people. They feel ashamed and embarrassed because of their acne lesions and tend to become withdrawn and introspective. They avoid social and sexual situations, abandon athletic activities, and become absorbed with themselves and their problems, frequently excoriating the lesions, removing comedones, squeezing pustules, and in general aggravating the already existing acne. Here again a vicious cycle is set up, and frequently it is difficult to differentiate cause from effect. It is our considered opinion that there is probably *no single disease which causes more psychic trauma, more maladjustments between parents and children, more general insecurity and feelings of inferiority, and greater sums of psychic suffering than does acne vulgaris.*

INFECTIOUS DISEASES

Infectious diseases, whether they are due to bacteria, fungi, viruses or other agents may perhaps be precipitated or perpetuated by emotional stress, either through the lessening of bodily resistance and lowering of the specific immunity, or by altering the local site so that the causal micro organisms can produce disease. The mechanisms by which the immunity and susceptibility are altered are unknown. The following diseases are representatives of dermatological infectious diseases which are known to be influenced by emotional factors.

Herpes simplex is a localized vesicular eruption due to a virus infection. This virus is probably a ubiquitous one, every ready to produce illness in the susceptible individual and site; menstruation, sunburn, windburn, intercurrent infections, fever, allergic or other reactions to foods,* drugs, and so forth, and emotional stress may precipitate this condition. Herpes simplex is therefore the disease in which the hydra-headedness of causes can most clearly be seen. Even though directly due to a specific culturable virus, artificially induced fever will elicit an attack in over 70 per cent of all persons—and if the fever were high enough and long enough in duration, probably close to 100 per cent would be affected (Keddie, Rees and Norman Epstein). Apparently, in a similar manner, emotional upsets can act as trigger factors in some

* The senior author has studied one case in which repeated attacks of herpes simplex followed ingestion of caviar; several in which such agents as peanuts, chocolate, shell fish, fish and iodized salt repeatedly elicited the herpetic eruption.

cases of herpes, and perhaps also in the related *herpetic stomatitis* or *canker sores* (aphthae).

Warts (*verrucae vulgares* and *verrucae planae*) are also, in all probability, due to a specific virus, but here the causal effect of emotional trigger mechanisms is not clearly seen. It is known that warts occur more frequently and are more numerous in children, appear almost in epidemic showers, and eventually disappear spontaneously. *The most important and most securely founded fact in all the maze of psychosomatic speculation is that 60 to 70 per cent of the cases of plane and vulgar warts respond to suggestive therapy.* This is the only instance in which scientific and statistical studies by a large number of independent authorities have unequivocally established an identical percentage of psychogenic cures of a visible and tangible somatic lesion—a lesion which is at the same time a tumor and probably virus infection (Br. Bloch, Bonjour, Memmesheimer and Eisenlohr, Allington, Sulzberger and Wolf, and others).

The cures seem to result irrespective of the type of suggestion, which may be anything from burying a peach pit under an apple tree, or a dead cat and "stump water," * to a placebo x-ray treatment or placebo injection. The exact psychological and physiological mechanisms which underlie the suggestion are not known. Here indeed is the ideal place for future objective studies on the relationship of psyche and emotions to somatic diseases.

Dermatophytosis is an infection due to various fungi which may be considered almost ubiquitous in the urban population of this country. Yet in spite of the continual presence of these fungi on our feet and in toenails and callouses, constant active infection is most uncommon. Activity occurs only when the systemic or local resistance is lowered. This lowering may sometimes be based on a relatively simple local effect, e.g., when the local site becomes macerated from excessive perspiration or from poor hygienic conditions. Probably for this reason, fungous infection is seen frequently in patients who have hyperhidrosis, exacerbations of the fungous infection sometimes fluctuating in chronologic relation and proportion to the hyperhidrosis. Since sweating itself fluctuates and is admittedly influenced by psychogenic and emotional states, it is not surprising to find that the exacerbations of fungous disease can be associated with the emotional changes. These remarks concerning dermatophytosis obviously apply also to other diseases associated with excessive perspiration, as *prickly heat* and "dyshidrosis," and "pompholyx." Thus, prickly heat, intertriginous eruptions, moniliasis, "athlete's foot" so-called vesicular dyshidrosis or cheiropompholyx and similar common skin diseases are prone to flare up, not only when the atmosphere is hot and humid, but also when the patient's emotions keep his skin sweating and hot, and "in a lather."

* Mark Twain.

Furunculosis is due to the staphylococcus group of micro-organisms, many of which are common invaders of the skin, but which do not usually gain a foothold unless a combination of conditions occurs to enhance their growth or to lower the resistance of the skin. The inter-related factors are obviously often varied and complex. The specific cause is the staphylococcus, but its pathogenicity and invasion may be influenced by or dependent upon the nutritional state of the patient, the metabolic-endocrine balance (e.g., diabetes), general physical state and immunological conditions, and the emotional state, which is related to all the aforementioned factors. In addition, the development of furuncles is undoubtedly largely dependent on local conditions such as friction, other trauma, maceration and more obscure local influences. The complexity of these various causes probably accounts for the common tendency to recurrences and new lesions, often despite topical medication. The patient must be studied and treated from all possible angles in order to achieve good results. In this disease also the possible role and mechanisms of psychogenic and emotional factors still await study.

MISCELLANEOUS DERMATOSES

There are many other dermatological diseases in which emotional factors may play an important part, but concerning which there are not as yet sufficient data. In this group, some would perhaps include exudative discoid and lichenoid chronic dermatosis of Sulzberger and Garbe,* dermatitis herpetiformis, Raynaud's disease, scleroderma, alopecia areata, and geographic tongue. However, there is no doubt that in almost any disease the mind and emotions play more or less important supportive roles in the cast of villainous characters. Therefore each school of thought is likely to have its own favorite list of diseases in which the psyche appears to hold a place somewhat nearer to center stage. This selection of diseases apparently depends "on the angle," or "where one is sitting" when the play goes on.

CONCLUDING REMARKS

In general, the clearly psychogenic dermatoses consist of self-inflicted and mechanically produced lesions, due either to manual or instrumental excoriation or to external application of damaging agents. These skin lesions may occur either in psychotic or severely neurotic patients and have been discussed in some detail.

Whether primary lesions are directly produced psychogenically is still questionable, but there is no doubt that emotional factors play an important part in the precipitation, exacerbation and perpetuation of certain dermatoses. The effect of emotional stress on the skin in the

* Sulzberger, Marion B and Garbe, Wm., "Nine Cases of a Distinctive Exudative Discoid and Lichenoid Chronic Dermatoses," *Archives of Dermat. and Syph.*, 36/2:217.

production of disease is indirect; in immunologic mechanisms it may, in some way, lower the threshold of reaction to the specific antigenic and allergenic agents, living or dead. In this manner the psychic and emotional states apparently can alter or disturb both systemic and local functions in such ways as to alter or interfere with local and systemic immunity. In some dermatoses, such as psoriasis, seborrhea, exudative discoid and lichenoid dermatosis, dermatitis herpetiformis, atopic dermatoses, and many forms of pruritus, even when no information is available as to the actual etiology, it can be recognized that some exacerbations are related to emotional stress and that the course of the disease may be influenced by emotional conditions.

On the other hand, *great danger exists in attributing to preponderantly psychogenic causes dermatologic diseases which are in reality due primarily and principally to other mechanisms.* This danger is all the greater because so many dermatoses have themselves such profound and substantial effects upon the patient's personality, his psyche and emotions. It is particularly because of this that the *direction* of the influences and relationships between psyche and skin disease may be mistaken by the physician, unless he applies great knowledge and preserves a meticulous objectivity. Few things in medicine are more likely to damage patients, prevent proper treatment, and delay the advance of scientific progress than the attempts to treat diseases such as scabies or fungous infections or acne or seborrhea or contact-dermatitis, or hives, or drug or other allergies, from the purely psychiatric viewpoint. Unfortunately we have seen an ever-increasing number of patients with these diseases treated in just this manner. These errors tend to become ever more frequent as the public, lay and medical, comes more and more under the influence of exaggerated ideas regarding the role of the psyche in the production of disease. If he is weak and willing, the nonpsychiatric physician need no longer spend the time and money for objective clinical and laboratory investigation; he need have no more failures either in diagnosis or therapy. For what patient who comes to the general physician for a somatic disease is free of psychic and emotional stresses? Which of us is without his neurosis or his mental difficulties? The physician who is tempted to lean upon these universal findings may soon delude himself by the notion that whatever disease he cannot speedily diagnose and remedy by other means must be due to the mind and emotions, and need simply be referred to an appropriate psychiatric specialist.

More disastrous even than its effects upon the individual patient and individual physician are the effects of such an attitude upon the progress of medicine. For no new somatic mechanism, no new infectious agents, no new metabolic disturbances or other chemical or physical causes of disease will be discovered, no further progress made in biochemistry or biophysics or in any branch of nonpsychiatric medicine once it is accepted that all those diseases which cannot *today* be recog-

nized as due to a known physical or chemical aberration must have their origin in the mind.

In dermatology, in such diseases as eczemas, hives and atopic dermatitis (disseminated neurodermatitis), one sees this pernicious direction of thought making considerable headway. For this reason, we believe that there should now be a substantial reduction in repetitions of vague speculation, in case reports, in statements of chronologic relationships and coincidences, and a return to objective methods of fundamental scientific research. Here indeed many a dermatologic lesion offers unrivaled opportunities and should speedily be utilized to advance the knowledge of psychogenic mechanisms in somatic disease. What better opportunity is offered to fundamental and objective research than by the wart—to investigate the manner and the conditions and the intermediate mechanisms by which suggestive therapy and the psychic changes *do* produce the complete and unequivocal cures of these easily diagnosed, absolutely characteristic and accessible, circumscribed tumors due to virus infection? Or what better object for study can be found than recurrent herpes simplex or aphthae, and the exact manner in which the psychogenic factors apparently act in concert with the other influences which periodically lower the local resistance of a given site to the culturable, herpetic virus?

Despite the antiquity of the concept and the avalanche of recent case reports, the number of proven and verified facts on the role of emotional factor in skin disorders is very limited. Extensive and carefully controlled investigations, correlating detailed psychiatric studies, psychological testing, personality analysis, virus, bacteriologic, neurologic, immunologic, histologic and other *objective dermatologic investigations* will have to be made before any more definite statements can be uttered as to the role of psychic factors in many of the common but still enigmatic dermatoses.

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ALCOHOLISM

Its Nature and Treatment

HARRY M. TIEBOUT, M.D.*

INTRODUCTION

AN attempt will be made in the following pages to supply some background information about alcoholism in the hope that, armed with this knowledge, the medical man may be better equipped to meet his responsibilities when confronted by the alcoholic problem. Only recently has the doctor felt the need for this sort of enlightenment but, with the movies, newspapers and magazines flooded with articles emphasizing that alcoholism is a disease, the man in practice finds himself acutely conscious of his limitations though facing many demands for help. It may be a disease to the alcoholics but to the doctor it is a bothersome problem and he fervently prays merely to be left alone. The times have changed, however, and whether he likes it or not, he will be consulted about the problem. For his own peace of mind, he should develop a point of view which will free him from too disturbing a sense of inadequacy.

In order to provide the looked-for information, the writer plans to summarize current thinking of the workers in the field. Admittedly such thinking has not yet jelled into many points of agreement; there has not been enough time for that. But certain areas of accord are now visible, enough in fact to furnish some degree of assurance to the individual seeking knowledge. Moreover, where it is possible briefly to present discordant points of view, that will be done also in the belief that such discussion will afford additional enlightenment.

DEFINITION

By definition alcoholism may be said to be present when an individual drinks compulsively. Obviously the key word in that definition is "compulsively." Its meaning thus becomes the kernel of the present-day approach to alcoholism and demands careful elucidation.

Compulsive behavior results from an inner drive which pushes the individual in the direction of certain acts even though he may be thoroughly aware of the foolishness or senselessness of what he is impelled to do. The average medical reader will recall how, in his student days, he was shown strange individuals who seemed to act normally enough about most things but who complained that they were bothered by a compulsion to wash their hands far more than ordinary cleanliness required. They knew it was silly but they couldn't help it; it was as if something inside made them do it. Asked what that something

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was, they groped for words but could make only one point quite clear, namely, that they could not rest comfortably until they had carried out the act which they felt inwardly compelled to do. No one who talked to those people would question the strength of the forces within them that produced the behavior nor that these forces arose from sources completely outside the reaches of the conscious mind.

The parallel with drinking holds true. Some drinking, in fact, most drinking, like most hand-washing, is within normal limits and can, therefore, be considered free from any compulsive element. But for a certain percentage of drinkers, the compulsive feature enters and, when it does, it produces alcoholism.

For the sake of accuracy, it should be stated that the parallel between the compulsion to drink and the compulsion to wash hands is not complete. The example of hand-washing was used to illustrate the nature of compulsive forces, not to say that alcoholism is an obsessional state. Perhaps more correctly it can be said that, in alcoholism, the compulsion is rather the development of uncontrollable urges for which alcohol serves as a form of relief. It is a type of self-medication.

A beautiful illustration of compulsive drinking occurs in one of the more recent books on alcoholism, Mrs. Natalie Scott's "The Story of Mrs. Murphy." The first drinking bout in the book occurs after Jimmy Murphy, the alcoholic, has killed the family's pet parrot in a fit of temper, after one year of successful sobriety. Horrified by his action, upset by the anticipated reactions of the family, particularly his father, Jimmy leaves home and begins a binge from which he does not emerge for over a year. The emotional disturbance set off by the demise of the bird has kindled the always latent tendency to drink. The compulsion then takes over.

Another example from real life took place in a person who had suffered an indignity for which there was simply no redress. She had to take it and, seething under the injustice, she went to bed and tried to fall asleep. But she couldn't and she heard the clock strike midnight, one and then two o'clock. Suddenly a bottle materialized at the back of her mind, as real and visible as any in broad daylight. And then the bottle slowly moved around to the front of her mind and she could feel herself wanting to grab it, yank the cork out and guzzle the contents. She then added: "If it had not been two o'clock in the morning, at that point I know I would have gone downtown," and spoiled, she could have added, the record of nearly a year. Although her compulsion did not lead to drinking, the compulsive pressure was there and almost certainly under other circumstances would have led to trouble.

DIAGNOSIS

Manifestly not all drinking bouts are started by such dramatic episodes; far more frequently the dividing line between ordinary and

compulsive drinking is extremely difficult to draw. The difference between wanting a drink and being compelled to drink is pretty subtle and depends on when a 'want' is transformed into a 'must.' The man who drinks regularly and has no compunctions about so doing is in no position to decide whether his drinking is compulsive or not. Since he consistently yields to the impulse to drink, he never tests the force of the drive behind the impulse. Only when the thought of a drink enters his mind and he resists it, can he appreciate the extent of the pressure behind the idea. If, despite a full realization that he should not touch a drop, he succumbs and takes a drink, then and then only can he realize that he is under compulsion and should take heed.

In other words, diagnosing the presence of a compulsive element is simple when the circumstances are sufficiently clear and striking. When the drinking, however, is part of the routine of one's way of life, then spotting the compulsive features is much more complicated. Not every Saturday night binge is compulsive but many compulsives welcome Saturday nights because they afford a good excuse to get drunk. With this excuse they thereby avoid facing the fact that they were actually submitting to their own inner urges toward alcohol and thus they can keep face with themselves.

On the surface, compulsives and noncompulsives may behave alike during any one Saturday night bout except perhaps for a tendency of the former to get a little more drunk. Underneath, however, things are happening inside the compulsive which in time will change the picture of his drinking and differentiate him from the rest of the drinking group. He will begin to have midweek drunks, or the Saturday nights will include Sunday and later part of Monday, or they will start Friday nights. In other words, the bouts will gradually grow in frequency, regularity, duration and quantity of alcohol consumed. Alcohol clearly is becoming more and more important to him. In the eyes of the outsider he is drinking more often and with worse results. And it is on the basis of this worsening picture that one can suspect the drinking is not of the relatively harmless Saturday night variety but has taken on a grim note of compulsion.

In summary, therefore, it may be said that compulsive drinking is disclosed either when the circumstances indicate the individual was reacting to inner pressure or when the history demonstrates that the drinking is steadily growing in frequency and severity.

The relationship between the two types of compulsive drinking is not well defined. They probably should be considered the far ends of a scale which measures the ease with which the origins of the inner pressures may be observed. At one end, the source is readily located, at the other it is completely obscure. In the intermediate spaces lie the various gradations between total obscurity and relatively complete insight. Actually the important issue at this point is not so much what caused the compulsive tension to arise as the fact that the tension did.

arise. Not everybody goes on a binge after killing a pet parrot—only the susceptible few. It is this factor of susceptibility to compulsive pressures which must be diagnosed.

ETIOLOGY

The origin of the compulsion in the alcoholic has not been located. Some investigators seek to find a physical explanation such as an allergy or sensitivity to the effects of alcohol or an inability normally to destroy it within the body itself. Others favor a psychological explanation. Probably a majority are still straddling, being unwilling or unable to commit themselves.

Fortunately the disagreement about causation is not too momentous. Since the accepted goal of treatment by all workers in the field is total abstinence, it really is not a matter of much immediate concern whether the defect is physical or psychological. Irrespective of cause, the alcoholic must avoid the first drink at all costs. If he does not, he will be in trouble.

One misconception perhaps needs to be done away with. Habitual drinking is not just a matter of sensory gratification, of liking the flavor and the glow; instead it serves a far deeper purpose, that of liberating the individual, temporarily at least, from the tension and the pressure of his inner conflicts and compulsions and transporting him to a place where troubles and distress may be forgotten.

A second misconception should also be corrected, namely the prevalent belief that excessive drinking is evidence of a weak will or character. Such belief ignores the compulsive element which, as already defined, is able at times to ride roughshod over will or character or whatever it is which keeps us on the right track. To talk to an alcoholic about will power is as sensible as to advise someone to lift himself by his bootstraps. It just does not make sense.

TREATMENT

Treatment is divided into three chief aspects: (1) the management of the acute effects of the intoxication; (2) repair of any physical damage inflicted by the combination of excessive intake of alcohol and limited intake of calories and vitamins; and (3) treatment of the underlying condition of which alcoholism is the chief presenting symptom.

Management of Acute Intoxication.—The handling of acute intoxication varies according to the amount of liquor imbibed, the frequency of its use and the period of time it has been resorted to. Obviously the person who has been on a week-end drunk offers a much different problem from the whisky-soaked individual who has been on a four months' bender. Simple sedation and putting to bed will carry the first patient through while the person who has been on a prolonged drunk needs much more thoughtful care.

His food reserves are depleted, his vitamins greatly reduced; his system is habituated to alcohol and he dreads the experience of withdrawal. Ideally he should be treated in a hospital where he can have the constant attention and supervision warranted by his condition.

Parenthetically, it may be pointed out that generally, after any long period of steady drinking, the patient is too weak physically to be noisy and a nuisance. He is by this time a sick man and a legitimate candidate for care in a general hospital. While admission may be properly denied to the person who is merely acutely intoxicated if only on the grounds of difficulty in control, no such justification exists when the man, having been on a prolonged bout, seeks hospitalization in order to end it. He is just as surely suffering from illness as is the pneumonia patient and just as rightfully should receive skilled care.

Whatever the place of treatment, certain principles must be observed. These have been discussed in detail in other articles, particularly in one by Lolli,⁴ to which the reader is referred for more complete information. Two schools of thought still exist about withdrawal. One group advocates immediate cessation as the quickest means of terminating the spree, often with the somewhat naive hope that memories of the suffering undergone will act as a deterrent to future drinking. The second group prefers more gradual methods in the belief that fewer risks are run and that possibly a more sympathetic approach by the physician will help to cement relationships. The argument between ruthlessness and appeasement has not yet been decided. The writer favors the latter program.

Regardless of the withdrawal procedure, sedation of some sort is necessary. Intravenous administration of 50 cc. of 33 $\frac{1}{3}$ per cent glucose with 10 to 15 units of insulin twice daily is routine in many places. It is given until the acute symptoms have subsided. Barbiturates and paraldehyde are useful but the danger of their being substituted for alcohol must be guarded against, particularly if the individual bears states of tension poorly.

Forcing fluids is often indicated for dehydration. Juices, vegetable or fruit are usually well tolerated and provide an acceptable medium for fluids as well as for caloric and vitamin content. Patients with persistent emesis generally gain relief from an intravenous injection of 500 to 1000 cc. of 5 per cent dextrose with isotonic solution of sodium chloride.

Acute attacks of delirium tremens with exhausting hyperactivity and wakefulness respond to intravenous injections of 7 $\frac{1}{2}$ grains of sodium amytal. Further dosage may be necessary to reduce the activity and produce sleep.

Repair of Damage Inflicted.—Restoration of vitamins should begin at once. Since alcohol impairs the absorptive capacity of the stomach, for the first weeks vitamins should be administered intravenously or intramuscularly. After that the avenue of administration is optional.

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Repair of Damage Inflicted.—Restoration of vitamins should begin at once. Since alcohol impairs the absorptive capacity of the stomach, for the first weeks vitamins should be administered intravenously or intramuscularly. After that the avenue of administration is optional.

One ampule of any of the standard preparations of vitamin should be given three times a week for at least the first two patient's condition determining how intensively the treatment be maintained. It is doubtful if much is gained except on a digestive level by prolonging doses beyond six weeks, that is, if the patient is eating normally and there are no residual effects of the former avitaminosis such as neuritis or skin changes. In such circumstances there may be some value in continuing high intake over a longer period.

The liver complications, acute hepatitis and cirrhosis, require supervised treatment which can be best administered only in hospital.

Psychological Approach.—The management of the acute phases, the restoration of damaged function, are relatively simple as contrasted to the complexities of trying to rid the patient of his alcoholic tendency. Deeply ingrained in the reactions of the patient, it can be removed by shouts or threats or scoldings. Only experience and training fit a therapist for the major task of freeing the patient from his bender. Such a task, obviously beyond the scope of the medical practice, still leaves the practitioner with the need to know what the task can be done and how to get the patient to the place or people qualified to do it. The doctor must do some steering, and unless he does it skillfully he will meet resistance which will prevent the patient from turning to assistance of any kind.

Resources for meeting the problem are now more plentiful. Psychiatrists are better equipped than formerly to tackle the job of rooting out the difficulties. Alcoholics Anonymous, an organization of ex-alcoholics with a 50 to 75 per cent claim of success, now has groups scattered all over the United States and Canada and is most willing to cooperate with the medical profession in helping their fellow victims.

Certain institutions have become interested in developing an aversion to liquor by means of a conditioned reflex. Although the physical aversion created is not a permanent solution it does seem to afford a breather, a period of time during which the individual can work out a new adjustment while free from the interference of alcohol. If no new adjustment is reached he will revert to his former habits.

For those who can pay for it, sanitarium care is often a necessary step, not only as a means of interrupting the drinking cycle and rebuilding the somewhat shattered physical structures but also, and more importantly, as a period for rest and relaxation. This relaxation causes the previous tense, nervous state to give way to a much calmer, more reasonable frame of mind in which alcohol is not so likely to be sought for comfort. Often, moreover, while in this relaxed condition, the individual is more responsive to discussions of his problems and is more likely to gain some insight into their causation.

For the less fortunate financially, a period on a farm away from the temptations of town frequently serves much the same purpose except for the absence of psychotherapy.

Recently a new resource has been added to the list. Special clinics for alcoholics, patterned after the pioneer one set up at Yale, are being established here and there throughout the country, staffed by a psychiatrist, an internist, and a social worker, all especially equipped to treat the problem from their particular angle. Such a clinic is a logical development since, like an allergy clinic, it concentrates different technics upon one single ailment and thus insures growth in the necessary knowledge and experience to combat it.

The physician's responsibilities do not stop when he guides a patient to adequate sources of help. He also must do what he can to induce in the patient a feeling that such help is necessary to his welfare. Unfortunately the alcoholic is by nature peculiarly endowed to turn a deaf ear to warnings, and so the physician may soon find that his well-meant advice is blandly discarded. Here the doctor must be patient and bide his time with two things in mind. First, he may get the alcoholic in a weak moment and persuade him to go to some place or some person for help. Second, he may in time be able to show that the drinking is creeping up on the patient in frequency, intensity or regularity and that the trend, if continued, will soon engulf him. Dangers to the physical body seem never so terrifying as threats to the individual's mental soundness and personal integrity. If and when he senses that he, as a person, may dissolve in alcohol, he is more likely to turn and seek help.

SUMMARY

Alcoholism is now considered a problem which comes in the domain of medical practice. There has been set forth some current thinking about the condition and its treatment. The role of the general practitioner in meeting the problem has been discussed.

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PSYCHOTHERAPEUTIC ORIENTATION FOR THE GENERAL PRACTITIONER

S. MOUCHLY SMALL, M.D. *

THE general practitioner is constantly faced with problems that require psychiatric understanding. He is usually the first to see the patient whose emotional conflicts have led to the development of a wide variety of physical and mental complaints. In many communities it is the general physician himself who must recognize and manage the majority of psychologic problems. It is essential, therefore, that the physician have an understanding of the role that emotions play in the production of the more common types of psychiatric problems that he is sure to encounter in his practice.

There is still a tendency among general practitioners to avoid the diagnosis of an emotional disorder. This attitude exists despite the fact that well over half of the patients who call on the physician do so because of problems relating directly to emotional conflicts. Many physicians prefer to enter upon prolonged physical and laboratory studies to the exclusion of attempts to understand the patient as a person who is subject to various emotional and environmental stresses. Some doctors, when finally faced with the lack of positive physical findings, will generalize to the patient that he has some toxic condition or inflammation of one sort or another. The rationale for this procedure is based on the assumption that the patient needs to feel that there is something physically wrong with him. It is true that this may give temporary relief, especially when combined with strong suggestion through the use of placebos. However, what usually occurs is that other symptoms appear in place of those originally experienced. This is inevitable since the essential psychologic problems remain and express themselves in one form or another. The new symptoms may be more refractory to therapy and at times may even be more incapacitating to the patient.

Even where the physician is aware of the importance of emotional factors he may feel that he cannot tell the patient that his or her complaints are psychogenic in nature. This attitude of misguided oversolicitude leads to further trouble. What particularly disturbs a patient is the statement "There is nothing wrong with you," made in the face of obvious alterations in function or distressing complaints. Most of these patients much prefer an explanation depicting the true state of affairs, presented in a manner acceptable to the individual personality. This is far preferable to a vague physical diagnosis, which

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is intuitively recognized as not the basis of the difficulty. In such situations, following adequate physical and laboratory examinations, it is desirable to review with the patient all that has been done and point out how the results of the several examinations indicate the absence of gross somatic disease. Then, instead of allowing the patient to remain perplexed and bewildered, the physician should try to build a foundation on which an understanding of the symptoms may be constructed. This may be accomplished through a discussion of common examples of the interrelationship between bodily functioning and various emotional states as seen in the average person, such as tachycardia with fear, vomiting with disgust, and headache with tension, using appropriate examples for the particular patient's complaints. Subsequently, in those patients who appear receptive to further insight, some of their psychologic problems may be carefully broached. If the patient is able to tolerate this previously suppressed (*consciously avoided*) or repressed (*unconscious and involuntarily forgotten*) problem, the discussion may then be amplified to provide a bridge to a better understanding and acceptance of his own symptoms as being related to unhealthy emotional patterns.

APPROACH TO THE PATIENT

The basis for a constructive therapeutic approach begins with the very first contact between the patient and the physician. It is the latter's behavior and attitude, even more than his words, that is a primary determinant in the evolution of a healthy, utilizable relationship or rapport between the practitioner and his patient. The sick person must feel that his physician is "all for him," that he is an individual who merits every consideration and not just another case to the doctor. People who are ill feel the need to depend on someone who can help them, someone who will not be impatient or intolerant. Destructive criticism will only lead the patient to suppress important facts without which his illness cannot be understood. If the patient is impressed with the practitioner's sincere interest in helping him and is encouraged to present his problems adequately, he will unconsciously lead the physician to significant sources of his difficulties. Do not moralize or judge the patient but listen earnestly and attentively. When it is necessary to ask highly personal questions the physician should not act apologetically for, if he does, it will only serve to embarrass the patient. Asking for this information in an open, frank and casual manner will automatically encourage responses given with a similar attitude. As a rule, it is wise to start with general and innocuous material in the history. After the patient comes to the realization that the doctor is there to help and is keenly interested in him, more sensitive topics may then be approached.

Many physicians will agree that the approach to the patient, as described, is desirable but that it is absolutely impossible in a busy

practice. They complain that they rarely, if ever, have sufficient time to go into the emotional background of a patient's difficulties. Yet, despite this, an occasional patient will remark that a certain well known, busy practitioner "makes you feel as though you are his only patient, that he has loads of time, and is interested only in you, even though he has a waiting room full of patients." Actually this doctor does not give more time in the long run to each patient than does another physician. His success in giving the patient this feeling lies in his attitude of interest in the patient, together with attention and an unhurried and leisurely appearance. Furthermore, it is the way he budgets his time, giving longer but fewer appointments, that enables him to achieve this excellent rapport with his patients. With such a relationship his suggestions will be followed more carefully and his therapy will be more effective and lasting.

Following the intimacy of the physical examination it is wise to give the patient another chance to talk, for he then may feel closer to the doctor and the information he vouchsafes may be more directly pertinent. In those situations where it is impossible to give a longer period of consecutive time an attempt should be made to conclude the appointment with a tentative formulation to the patient so that he is not left dissatisfied or confused. The physician *summarizes the findings* as far as he has gone and indicates the need for further study and talking. A premature final opinion will destroy the patient's confidence, for he may feel that it is based on a hurried and inadequate examination.

The diagnosis of emotional causation does not mean the necessity of referring the patient to a specialist. There are large numbers of problems of a minor nature which the general practitioner can and should manage by himself. At times he may require the assistance of a single consultation by the psychiatrist, but for the most part he should be able to continue the treatment recommendations as given by the consultant.

With a psychologic orientation the general practitioner can differentiate between those cases in which he may utilize simple psychotherapeutic measures with success and those which require care by a specialist. Fortunately, relatively few psychotics are seen in daily general practice compared with the large numbers of neurotic individuals who confront the physician. Many in the latter group can be helped by supportive or symptomatic therapy combined with psychologic insight into their troubles.

In the realm of problems that can be helped by the practitioner are those patients who have mild or transient neurotic symptoms in which environmental factors predominate as the immediate cause. Patients with mild anxiety symptoms with characteristic physiologic changes, persons with marital problems which are the result of lack of knowledge or understanding, or patients reacting to adverse changes in their

is intuitively recognized as not the basis of the difficulty. In such situations, following adequate physical and laboratory examinations, it is desirable to review with the patient all that has been done and point out how the results of the several examinations indicate the absence of gross somatic disease. Then, instead of allowing the patient to remain perplexed and bewildered, the physician should try to build a foundation on which an understanding of the symptoms may be constructed. This may be accomplished through a discussion of common examples of the interrelationship between bodily functioning and various emotional states as seen in the average person, such as tachycardia with fear, vomiting with disgust, and headache with tension, using appropriate examples for the particular patient's complaints. Subsequently, in those patients who appear receptive to further insight, some of their psychologic problems may be carefully broached. If the patient is able to tolerate this previously suppressed (*consciously avoided*) or repressed (*unconscious and involuntarily forgotten*) problem, the discussion may then be amplified to provide a bridge to a better understanding and acceptance of his own symptoms as being related to unhealthy emotional patterns.

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life situations by mild depressive illnesses may be helped considerably through simple technics such as reassurance, suggestion, explanation and education, with supportive medication used as a temporary measure. Oftentimes the course of a structural disease process may be sufficiently modified through the use of psychotherapy to the point where the intensity and duration of symptoms are markedly reduced. This is especially true in the psychosomatic disorders where emotional problems are directly related to the onset and exacerbations of the disease.

It is advisable for the practitioner to know something about neurotic mechanisms and symptoms but this knowledge should not be used indiscriminately in uncovering inner conflicts, for it may do considerable harm if given at a time when the patient cannot accept it. Patients with long-standing or severe neuroses or those with psychotic reactions should be referred to the psychiatrist. While the general practitioner will often treat certain delirious patients or those with organic psychoses, a consultation with a psychiatrist may prove fruitful in that management of neurotic patients is made easier. Those patients who are deeply depressed and express self-deprecatory ideas are often potentially serious suicidal risks. If these symptoms are accompanied by persistent insomnia, anorexia with weight loss, decrease in sexual libido, or menstrual disturbances, the self-destructive danger is even greater. It is a fallacy that those people who talk about suicide never attempt it. They are the very ones to watch carefully.

CASE HISTORIES

A few examples of the type of case that can be managed successfully by the general physician will now be given. In all of these patients, the symptoms disappeared or were controlled by the use of relatively simple forms of psychotherapy such as explanation, education, reassurance and slight modification of the environment.

CASE I.—A. L. was referred to his general physician because of severe disabling abdominal pains. Physical examination revealed only some mild tenderness on deep palpation in the midepigastrium. His pains came at irregular intervals and were occasionally, though not always, relieved by milk or alkalies. A gastrointestinal series revealed hyperperistalsis and a moderate degree of pylorospasm. At first there was some question about a duodenal ulcer but repetition of the x-ray series divulged no indication of any peptic ulcer. His physician prescribed a mild sedative and this seemed to give more relief than anything else that had previously been administered. When it was suggested that his symptoms might be related to some emotional difficulty the patient readily agreed, stating that he had "sort of had the same kind of feeling." He came for his psychiatric consultation in a most friendly and cooperative manner.

During this interview he reviewed the story of his present illness essentially as he had already told it to his own physician. He was 32 years old and had been married for four years. There were no economic worries, socially they were accepted by the others in the small community in which they lived and his sexual adjustment posed no problems. The only new situation that preceded the onset of his symptoms was the fact that his sister-in-law had come to live with them.

He did not make any further remarks about her until the chronologic relationship between the onset of his abdominal pains and her coming to live with them was pointed out. A. L. was asked to tell me something about her. He started by saying that she was a young girl of 16 years of age, attractive, popular and friendly, but she had the bad habit of staying up late. When asked why this bothered him he added that their bedroom was right next to the kitchen where she often sat and read and that she also had the pernicious habit of walking into their bedroom without knocking even though the door was closed. Then followed a long story of how his wife refused to have sexual relations while the sister was still awake because she might walk into the room and that by the time she had gone to bed he was usually fast asleep. He worked hard as a salesman and he felt worn out the next day unless he had a full seven hours of sleep. He recalled how he lay awake one night tossing and turning, unable to fall asleep and growling progressively more angry at his sister-in-law. It was shortly thereafter that his pains began to occur quite regularly.

For the first time during the interview he appeared to relax as he saw the connection between his unexpressed anger and resentment and the spasms in his stomach. He was urged to tell his sister-in-law that her habits were interfering with his sleep and to suggest that she read in her own bedroom. In addition it was suggested that he lock his bedroom door when marital relations were contemplated. This seemed to satisfy his wife who no longer had any objections under these circumstances. These recommendations were reinforced by his general physician who enlisted the support of his wife, and this resulted in a complete relief of the patient's symptoms.

Comment.—With this particular patient the key to the problem was obtained when he was asked if there had been any changes in his family relations, occupational adjustment or financial status prior to the onset of his symptoms. His answer was in the negative but he did casually mention the fact that his sister-in-law had come to live with them at that time. At first he did not attach any conscious importance to this circumstance and remarked upon it only in passing. However, when the time relationships were emphasized and he was specifically asked for more information relating to this change in living conditions he revealed its far-reaching significance. Subsequently the psychosomatic relationship between various gastrointestinal disturbances such as diarrhea, nausea, vomiting and spasms ("knots in the stomach") and emotions were explained to the patient. This closed the final gap in his recognition of the meaning of his abdominal symptoms. Once these dynamic relationships were discussed with his own doctor the latter was not only capable of managing the patient successfully but it also gave him insight into some of the wife's complaints.

It would be misleading to intimate that all cases of psychogenic gastric spasm can be resolved as easily as this one. This case is presented because of its relative simplicity and its excellent response to superficial psychotherapy in the form of simple manipulation of the environment, education and surface interpretation. However, most of these types of functional disorders do respond to a psychotherapeutic approach. It may be necessary to prescribe drugs, such as sedatives and antispasmodics, to give the patient some immediate relief from his symptoms. If this is not done he may seek another physician who will

life situations by mild depressive illnesses may be helped through simple technics such as reassurance, suggestion and education, with supportive medication used as a measure. Oftentimes the course of a structural disease be sufficiently modified through the use of psychotherapy where the intensity and duration of symptoms are mild. This is especially true in the psychosomatic disorders where problems are directly related to the onset and course of disease.

It is advisable for the practitioner to know some of the mechanisms and symptoms but this knowledge should not be indiscriminately in uncovering inner conflicts, which can do considerable harm if given at a time when the patient is not ready. Patients with long-standing or severe neuroses or who have had previous actions should be referred to the psychiatrist. The practitioner will often treat certain delirious patients with psychoses, a consultation with a psychiatrist is advised. In the management of neurotic patients is made more difficult if they are deeply depressed and express self-destructive tendencies with serious suicidal risks. If these symptoms include persistent insomnia, anorexia with weight loss, decreased libido, or menstrual disturbances, the self-harm is usually greater. It is a fallacy that those people who do not attempt it. They are the very ones to watch.

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CASE I.—A. L. was referred to his general physician because of abdominal pains. Physical examination revealed on deep palpation in the midepigastrium. His pains could be relieved occasionally, though not always, by milk and sugar. X-ray series revealed hyperperistalsis and a moderate degree of distention. There was some question about a duodenal ulcer. No indication of any peptic ulcer was found. The patient and this seemed to give more relief than any other treatment administered. When it was suggested that the emotional difficulty the patient read "the same kind of feeling." He came in a friendly and cooperative manner.

During this time he had already been married for 10 years. He accepted by adjustment of his symptoms.

SYMPTOM FORMATION

Rational psychotherapy is dependent on a psychodynamic understanding of symptom formation. Unless the general physician can see how symptoms evolve and spread within the personality he is likely to become routine and superficial in his approach and then when the patient does not respond favorably to his treatment he may become resentful and refer to the patient in a derogatory manner as a neurotic. Actually designating a patient as a neurotic should be the starting point of treatment rather than the final straw in a rapidly deteriorating patient-physician relationship. To diagnose a patient as a neurotic is in itself a challenge to the doctor to find out what is disturbing the patient emotionally, how and why he developed his particular symptoms and what the illness accomplishes for the patient consciously or unconsciously. In short, the problem facing the physician is the question of the meaning of neurosis.

Unfortunately the term psychoneurosis often carries with it the idea that the patient's complaints are imaginary. This is patently untrue and misleading. Patients with neurotic vomiting do not imagine they vomit—they actually do so whether x-rays or gastroscopy do or do not show structural defects to account for it. A neurosis means that the individual is psychobiologically out of balance, that emotions have gone wrong and have upset the individual's homeostasis. The symptoms are in reality a reaction of the total personality attempting to re-establish a psychodynamic equilibrium. They may be conceived of as defenses or compromise solutions of conflicts between different parts of the personality.

The basis for a neurosis is *conflict*—conflict between wishes, desires and strivings on the one hand, and social restraints, cultural restraints, and inhibitions imposed by the particular mores of the community in which the person lives, on the other hand. The individual may be aware of these conflicts at times but more often they are less obvious and the individual may or may not be conscious of their presence. A situation which in itself may appear to be relatively insignificant may stir up an old conflict which was never satisfactorily resolved and in this manner gives rise to symptoms and emotional reactions far greater than the apparent value of the precipitating incident.

Neurotic symptoms vary considerably but for each patient they form a pattern which represents that individual's particular reaction. The form and type of symptom is influenced by the previous life experiences, behavior patterns and illnesses of both the patient and those about him. In addition it may be a symbolic representation of the conflict and as such it holds clues to the nature of the underlying problems of which the patient is not conscious.

How does a conflict get translated into a physical disability such as a paralyzed hand? What is the mechanism at work? We can only hint

procedure to give him symptomatic relief and is not a cure. Under these circumstances the practitioner may then proceed with an adequate investigation of the social, economic, sexual and emotional stresses in the patient's life. Any reassurance that is given should be only on the basis of facts established by the necessary physical and laboratory examinations. Interpretations should not be given prematurely for it may make the patient worse. Only when relationships are quite obvious should the practitioner reinforce it with the weight of his own authority.

The general physician has a definite advantage over the psychiatric consultant in knowing not only the patient but the family as a whole. This often enables him to understand psychiatric problems from a developmental point of view. He is in a position to see the importance of the early formative years in determining particular forms of behavior, habits and attitudes which are often carried over into adult life. For example, a mother who is oversolicitous in regard to bodily health may predestine her child to a hypochondriacal attitude toward various little pains and aches. The influence of such parental behavior is clearly shown by the following case.

CASE II.—Tim was an only child of 8 years of age. His mother was an attractive woman in her early thirties who rebelled against her present lot in life. As a girl she had been very popular, and her life had consisted of a round of parties, dates and social engagements, with little responsibility at home. When she married everything was fine until her son was born and she became burdened with the necessity of keeping house. Shortly thereafter she developed a duodenal ulcer. This she used effectively to get whatever she wanted from her husband—jewelry, a fur coat, and so on. When she was frustrated by not getting what she wanted her pains would increase and she would continually complain to her husband until the desired article was purchased. The boy apparently sensed the value of illness as a means of attaining one's desires. Some time later he met with a difficult situation in school and complained of stomach-ache. Accordingly he was sent home by his teacher. This occurred a number of times and subsequently it was noticed that after a short while at home he would resume active play, eat well and not be incapacitated in any way. His family doctor advised that the stomach-ache be disregarded by the teachers. This was done and after a while the gastric complaints disappeared, only to be replaced by severe headaches whenever he had to face any difficulty, such as an examination. The utilization of such a retreat into illness as a defense was obvious to the practitioner but he did not follow through with an adequate psychotherapeutic procedure. He was correctly oriented but did not go far enough. No effort was made to find out why this boy reacted as he did to examinations. When he came for consultation he was found to be somewhat dull, and a psychometric examination revealed him to have a borderline low normal intelligence. The private school he attended had high academic standards with which he could not cope. Furthermore his parents repeatedly emphasized the importance of high grades without taking his intellectual capacity into consideration. When the problem was formulated to his parents and the help of the school authorities was enlisted he was given additional help by a tutor, and the need for high scholastic achievement was deemphasized. In addition he was taught certain manual and physical skills at which he excelled. This led to an alleviation of Tim's complaints. However, unless his mother receives psychotherapeutic assistance it is likely that he will continue such neurotic patterns of response to difficulties.

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at some of the forces that are operative. The human personality characteristically shies away from unpleasant situations and puts off doing disagreeable tasks. We tend to think of the "good old days," not because they were particularly good but rather because unpleasant experiences are forgotten and not easily recalled. Thus we see that in addition to passive forgetting in which relatively unimportant matters like the content of yesterday's meals are forgotten, there is also the so-called active type of forgetting called repression. In this latter type of memory difficulty, unpleasant or disturbing emotions associated with a thought cause it to be automatically forgotten. Patients anticipating a painful time at the dentist's office may unknowingly (unconsciously) forget about the appointment until it is too late to keep it. In other words, the whole unpleasant idea, thought or situation is repressed into an unconscious area of the personality.

Repressed conflicts within the personality make for unrest and inner tension. These thoughts, ideas and wishes which are pushed back into the unconscious do not lie dormant but constantly strive for expression. These thoughts have emotion, feeling and energy associated with them which also seek liberation and expression. To keep these unacceptable thoughts and feelings from breaking out, defenses and compromises, which are often the symptoms themselves, may be necessary.

To get back to the problem of the paralyzed hand, we can now see that the individual probably has an intense conflict revolving around the use of the hand. In the case of a soldier it may be a conflict between a desire to be patriotic, to fight for his country as a brave man and an overwhelming drive for self-preservation. When he cannot resolve this painful conflict, the whole complex of ideas is repressed so that he is no longer consciously aware of its existence. However, the energy associated with this conflict remains active and strives for expression, and it is this energy which ultimately is translated into the symptom which keeps him out of active battle. If the hysterical paralysis should be drastically removed by hypnosis, then the complacent calmness which characterizes the patient's attitude will give way to intense anxiety which was originally associated with the repressed conflict.

A more detailed example of neurotic vomiting is the case of George L., to be described. Here the previous life experiences with father plays an obvious role in determining the form of the symptom. At first an attempt was made to carry out treatment through consultation with the general practitioner. However, when a depressive reaction set in more intensive psychotherapy by the psychiatrist was required.

CASE III.—His parents were extremely worried about George L. Until a few weeks before he had been an outstanding scholar at a large eastern university. Then, for no apparent reason, George began to vomit repeatedly and was unable to eat; his sleep was troubled, and he could no longer concentrate on his studies.

In a period of two weeks he lost over 10 pounds. His parents became more frantic when, after numerous tests and x-ray examinations, no physical disease could be found to account for his difficulty. The roentgenologist had said something about spasm in the stomach and remarked that such spastic conditions often were related to emotional problems.

George was the older of two boys. His younger brother, Henry, was two years his junior. George was blessed with a handsome physique and a brilliant mind and in these ways easily outshone his younger brother. He carried his father's favor primarily through his intellectual accomplishments since that seemed the only thing that could get father to give him the affection, interest and understanding he so desperately craved. Father's way of exhibiting his approval consisted primarily in giving material things rather than the time and interest George desired.

George's father was a self-made man. He came from a large, typically American family in the Western part of our country and by dint of perseverance and hard work he had finally become one of the top executives of a large company. The father was essentially practical and impatient with sentimentality, and indeed his entire life was governed by the need for accomplishment and the attainment of financial security and social prestige. However, it was not always so. More than fifteen years ago, when George was but 5 years old, the father had been sent on a mission to a small country in the Near East to conduct a survey of its oil-producing potentialities. He made extensive plans and finally arrived there with his wife and two children. Living conditions were intolerable. The amount of work that was necessary would take at least a year and he felt he could not live through that length of time in the oppressive climate and miserable living conditions that they were subjected to. After being there one month the father began to have recurrent bouts of nausea and vomiting. He was seen by a number of physicians who assured him that he had no intestinal disorder. Intuitively, George's father recognized the significance of his vomiting—the fact that he was rejecting this intolerable situation. He quickened his pace, drove himself day and night and miraculously accomplished the year's work in a period of six months and left for home. On his return to this country he quickly regained all his lost weight and all his symptoms disappeared. This experience further convinced the father that self-discipline in forcing oneself through situations was the only real manly way to handle any problem. This story was well known to George who had always been proud of his father's repeated promotions.

George's mother was a tender, sweet person who was swept away by her husband's driving ambition and finally succumbed, herself, in the form of a gradually increasing hypertension to the point where her physician warned the family not to upset her in any way. The little time she did have available was absorbed by her husband's social interests so that the two boys really grew up in an emotionally cold home where things were run efficiently but with little human warmth.

As though this were not enough, George had recently suffered a reverse in a love affair. He was very fond of a girl who seemed to be the direct antithesis of his family in the sense that she was loving, kind, affectionate, warm and seemed completely interested in his welfare and happiness. She was not a particularly attractive girl but her feeling for him made up for any other deficiency he discovered. The storm broke when he received an announcement of her engagement to another fellow. The following day his vomiting began.

After a few interviews with George it became apparent to the physician that he, like his father before him, was evidently unable to accept the situation and was rejecting an intolerable frustration. He felt he had been let down by the whole world. When the relationship between his vomiting, which mimicked his father's reaction, and the loss of his girl friend was pointed out to him his vomiting ceased, only to be replaced by crying spells for which he could not account. Just as he had regurgitated his food previously without apparent reason, now tears flowed without his being aware of the cause. He was now able to eat but nevertheless

was unhappy. It was only after many more interviews that George realized he was never really in love with the girl, that she only gave him the affection he had always wanted from his parents, that she had nourished him with the milk of human kindness which he had so sorely missed at home. When she left him he felt completely let down by everyone and rebelled against it unconsciously through the mechanism of vomiting.

Today, after many more hours of treatment, George is back at school—happy and doing better work than ever before. He no longer works to achieve high grades with the sole purpose of getting parental affection, but now works hard because of the love of his work itself. He is emotionally more mature and gets on much better with all his friends of both sexes.

DOCTOR-PATIENT RELATIONSHIP

The crux of *successful treatment* lies in the doctor-patient relationship. This is true whether the therapeutic approach is superficial and limited or deep and intensive as in an orthodox Freudian analysis. The physician must give the patient the feeling that he is sincerely interested in his welfare and will help him in whatever way he can. This feeling cannot be imbued in the patient solely by saying so. It must be repeatedly corroborated by the physician's every action, demeanor and attitude. It is only then that the patient will feel free to talk about things which may be painful or distressing or be willing to expose his innermost emotions and attitudes. Do not urge the patient to confess for this is attended by moral and religious implications which do not properly belong in a medical situation. The physician should not set himself up as a minister or judge; on the contrary his orientation should be primarily in terms of physical and mental health.

The opportunity to have an understanding and trained person listen to his various complaints (psychocatharsis) often gives considerable relief in itself. In addition, if the physician by virtue of his experience is in a position to allay many of his patient's unreasonable and unfounded fears such as fear of "going crazy" or the fear of imminent death, he has added much more to the individual's sense of emotional security. The facts of the physical and laboratory examinations may be used to reinforce the reassuring statements that the patient does not have the disease or outcome he dreads.

In the management of patients with neurotic symptoms the general practitioner should confine his therapy to simpler and more superficial methods than the psychiatrist. He must be aware of the danger of uncovering sensitive material before the patient can deal with it emotionally. Even though the psychodynamic factors may be quite clear to the physician it is important for him to use this knowledge judiciously and not divulge it prematurely. To do so may lead to a serious exacerbation of the symptoms, self-destructive impulses or, in a certain few cases, to a psychotic development. The practitioner who is not trained in analytic, dynamic psychiatry may find himself in the analogous position of the physician who, having opened the peritoneal cavity, finds a loop of gangrenous bowel whereas he expected to find,

and is only trained to deal with, an uncomplicated appendicitis. He may know what should be done but cannot deal with it unaided. In both the surgical and psychiatric situations the direction of a consultant is necessary.

However, this should not lead to an avoidance of a psychodynamic appreciation. Understanding the emotional conflicts involved in the formation of the patient's symptoms will guide the physician in his suggestions for simple modifications in the environment which may decrease tension and anxiety. It enables the doctor to direct his reassurance and constructive suggestions to the very point where it can do most good. Then he can follow through with encouragement to induce the patient to face his reality problems in a mature fashion.

One of the most valuable psychotherapeutic procedures is to demonstrate to the patient, through the use of his own associations, the meaning behind the symptom. When the doctor is able to show how vomiting is just another way of rejecting certain wishes concerning which there is conflict, the patient will often corroborate this and give additional valuable material. Such interpretations should be given only when there is abundant evidence to suggest it. The idea should be formulated in a subtle or oblique manner, perhaps even in the form of a question, so that the practitioner can see how the patient accepts it. If too much anxiety is stirred up by even the suggestion of a connection between the symptom and his emotions then it is folly to go further. This anxiety reaction constitutes a danger signal indicating that the patient is not yet ready to give up his neurotic defenses against his conflicts. Under these circumstances therapy should be directed toward building up the patient's self-respect and self-confidence. In other words, strengthening of the ego instead of trying to uncover conflicts.

There may be occasions when the physician senses that the patient is trying to run away from treatment. When this occurs it is helpful to indicate the ambivalent attitude toward the physician and his therapy to the patient. The patient wants to be helped with his distressing symptoms and comes to see the doctor because of them. Yet when he is asked to talk about himself, the thoughts which come to mind may be too painful or upsetting. Thus the actual treatment situation itself becomes a conflict. If this natural reaction is explained to the patient he may feel more comfortable about expressing his resentments and appreciation.

This is illustrated by a woman who suffered with obsessional thoughts associated with anxiety. She originally went to see her doctor because of cardiac palpitations. When a careful physical examination revealed no explanation for her symptoms he inquired further into her difficulties. Under the influence of his sympathetic, listening attitude she poured out things about her past life which she had never revealed to anyone else. Following this she was unwilling to keep her

spinal fluid examination as a *sine qua non* for the diagnosis of neurosyphilis.

If the spinal fluid tests are indispensable for the recognition of the syphilitic involvement of the central nervous system, do they have the same importance for the evaluation of therapeutic success or failure? Here again we have to stress that clinical signs and symptoms are by no means always helpful. Everybody knows that sometimes irreversible destruction of nervous tissues has occurred before treatment was started and that, therefore, we cannot expect a complete recovery even with ideal therapy. On the other hand, the syphilitic process may continue even though the patient improves, as in general paresis where patients may have temporary remissions, with remarkable clinical improvement, only to relapse later. It is obvious, therefore, that to appraise the effect of treatment in neurosyphilis we must avail ourselves of a standard which mirrors the activity of the syphilitic process in the central nervous system in a more reliable way than does the clinical status. Based on our extensive experience in this field we firmly believe that spinal fluid tests serve this purpose in an excellent manner. They do it, however, only if properly performed and interpreted. At least four different tests must be performed on the spinal fluid of patients suspected of having neurosyphilis. These tests are so important in diagnosing neurosyphilis and evaluating the results of treatment that they demand some discussion here.

Complement Fixation Test.—The first test for neurosyphilis is the complement fixation test. Specific tests for syphilis have been greatly refined in the past decade. Cardiolipin has been introduced as antigen. This antigen constitutes chemically known substances which have rendered the test more specific. In addition, the quantitative complement fixation tests,¹³ as performed by the New York State Department of Health Laboratories, afford a superior means of determining the amount of reagin in spinal fluid. Through the courtesy of Dr. E. R. Maillard we have been able to obtain such quantitative tests for our patients treated at Bellevue Hospital.

The presence of reagin in the spinal fluid indicates, with few exceptions, a past or present syphilitic infection of the central nervous system but it tells us very little about the activity of the process. Fortunately, in the case of the spinal fluid we have other tests available which do give us a fairly accurate picture of the status of the infection.

Cell Count.—The most important of these additional tests is the cell count. The first sign of a syphilitic invasion of the central nervous system is an increased cell count; an active inflammatory process due to syphilis is rarely present with a normal number of cells in the spinal fluid. Authorities have differed over the upper limits of normal cell counts in the spinal fluid. Using the 10 cu. mm. chamber of Nageotte, most French investigators considered 10 to 15 cells in 10 cu. mm. as the upper limit of normal. Fuchs and Rosenthal in Vienna, who introduced

the chamber carrying their name, holding 8 cu. mm., believed that from 0 to 2 cells in a cu. mm. was the normal standard. Later careful studies, especially those of Neel¹⁶ on thousands of spinal fluids, proved beyond doubt that more than 8 cells per cu. mm. constitutes a deviation from the normal. We concur with his experience and consider up to 8 cells per cu. mm. (9/3) within normal limits, 8 to 5 cells (9/3 to 15/3) as borderline and more than 5 (15/3) cells as definitely pathologic. If the pleocytosis is a criterion of activity of the syphilitic invasion of the central nervous system, then it ought to disappear once the inflammatory process has been checked. This invariably occurs when the active syphilitic process has been arrested. There is universal agreement among all observers that following adequate treatment for neurosyphilis there is rapid reversal of the abnormally high cell counts to normal values. We, therefore, feel entitled to assign a paramount importance to a reliable cell count.

Total Protein Estimation.—Another test of the spinal fluid which may give a clue to the nature and tendency of the pathologic process within the central nervous system is the total protein estimation. Again in general medicine there is no doubt about the significance of increased protein values of the spinal fluid. The difficulty in the past with total protein determinations has been the inaccuracy of the methods used. Fortunately in the last few years there became available an electrophotometric apparatus based on a selenium cell, which measures the turbidity of the spinal fluid caused by sulfosalicylic acid in such an exact manner that values obtained by it can be duplicated without difficulty. Now that reproducible figures for total protein estimation are at our disposal, it is possible to observe the changes over long periods of time and, therefore, to draw conclusions as to the trend of the process. Patients whose syphilitic disease in the central nervous system has been definitely checked by treatment exhibit a gradual decrease in protein, whereas patients who relapse after treatment present increasing amounts of total protein on repeated tests.*

The three spinal fluid tests so far discussed give us only information about the specificity, intensity and activity of the syphilitic process. In addition, we would like to know whether the infection involves primarily the interstitial tissues or the parenchyma proper of the central nervous system. This information seems important since it is customary to classify neurosyphilis in meningo-vascular or parenchymatous forms; the latter are considered more dangerous and require more vigorous methods of treatment. This differentiation is based on a pathologic-anatomical point of view. Few clinicians, guided solely by signs and symptoms, are able to determine positively in many cases what principal structures are involved. This is proved by the fact that even among experts there is frequently considerable doubt as to whether a clinical syndrome is caused by a purely vascular, meningo-vascular or parenchymatous involvement. This is understand-

able, because, as Merritt, Adams and Solomon¹⁴ point out, "these clinical entities are not mutually exclusive clinically or pathologically, since it is rare for only one of these elements to be involved in a given case."

Colloidal Gold Tests.—The colloidal tests, especially Lange's colloidal gold test, seem to provide a somewhat guarded answer to our question as to the prevalent nature of the neurosyphilitic process. Experts in this field claim that it is specific globulins, the gamma globulins, which act as a precipitating agent in the colloidal tests. These specific agents appear in the spinal fluid when parenchymatous structures of the central nervous system are being destroyed. They are responsible for the so-called paretic curves (first zone). If there is involvement of only the interstitial tissues (meninges and vessels), then albumins, mostly deriving from the serum because of the increased permeability of the cerebral vessels, penetrate into the spinal fluid. They exert a protective action on the colloidal sols, so that the resulting curve in the colloidal test is either a mid-zone or end-zone curve, thus indicating a milder form of neurosyphilis.

Until recently, it was difficult to obtain sensitive colloidal solutions which would give reproducible results when used at different times on the same spinal fluid. Through the ceaseless efforts of Lange¹¹ it has become possible to prepare a gold sol which fulfills all the requirements of a dependable test substance. Using a standardized color scheme, which comprises values from 0 to 20, the new Lange colloidal test gives constant color changes which can be duplicated and which afford an accurate comparison of results. We, therefore, now have at our disposal not only a test which gives us a clue to the kind of process within the central nervous system, interstitial or parenchymatous, but which also furnishes us quantitative information by adding the sum total of the color values. The color numbers in the 10 tubes should not total more than 45 in a normal fluid. The highest possible sum total would be 200.

Further Comments on the Importance of Spinal Fluid Examination.—We consider these four tests as obligatory. Each one represents a totally different and independent approach to the study of the pathologic process in the central nervous system. But the results of these tests taken together form a syndrome whose interpretation as a whole affords reasonably accurate information about the syphilitic process in the central nervous system.⁴ Whereas there is no parallelism between the clinical syndromes and the status of the neurosyphilitic process, there is a close and constant relationship between the spinal fluid syndromes and the trend of the infectious or degenerative involvement of the central nervous system. If there is activity, then we observe pathologic cell counts, and in the course of time increasing quantitative values of protein estimation, colloidal gold reactions and complement fixation tests. If, however, the process is abating or comes to a standstill, we have correspondingly normal cell counts and decreasing

values in all the other tests. It is noteworthy that in the latter case the cell count is usually the first to give normal standards, just as it is the first to be high at the beginning of the infection of the central nervous system; then follow the protein values and finally colloidal gold reactions and/or Wassermann reagins in unpredictable time intervals.

It may be surprising that preliminary to the discussion of the latest methods of treatment of neurosyphilis so much stress has been laid on the spinal fluid tests. But whoever is confronted with the task of evaluating the results of any type of therapy in neurosyphilis must know how difficult it is to measure the success or failure of treatment if one is dependent solely on the clinical manifestations. No one will deny the importance of clinical signs and symptoms for the diagnosis of neurosyphilis. But since many of these may be permanent in nature as, for example, pupillary changes, loss of deep tendon reflexes and severe hemiplegia, it is plain that they cannot give a clue to the activity of the syphilitic process. Still less can they serve as a guide for the estimation of therapeutic effectiveness.

As mentioned in the beginning, the parietic may show definite improvement of his mental status and may nevertheless continue to harbor his dangerous infection. The tabetic may retain his lightning pains or gastric crises and yet the syphilitic process may have been arrested. We have to keep in mind that the optimum result we can achieve in treating an infectious disease is to eradicate the noxious agent as soon as possible and without harm to the patient. What is left in form of structural damage after we have succeeded in this effort, is beyond the scope of specific therapy. As much as we may wish to obtain the maximal clinical benefit, we are impotent beyond our effort to kill the last spirochete. This is one of the least understood facts in the management of neurosyphilis. The diverse clinical signs and symptoms we encounter in our dealings with neurosyphilis cloud the issue. We are prone to forget that the principal cause of all the protean manifestations of the syphilitic disease is the *Treponema pallidum* and that the manifold clinical syndromes are only the response of the host to the infection. Why this answer should vary in such a bewildering manner is a problem for future research. At present we cannot do more than arrest the process and hope that the restorative power of the human organism will do the rest.

RESULTS OF TREATMENT

We now feel adequately prepared to enter into the discussion of our main topic, the latest methods of treatment of neurosyphilis. There can be little doubt that penicillin is assuming top rank among them. The astonishing results obtained in the treatment of early syphilis with penicillin immediately suggested its use in neurosyphilis. Therapeutic success was anticipated in those types of neurosyphilis which were

known to respond well to the older forms of arsenical and heavy metal therapy. Very soon, with few exceptions, there was general agreement among all investigators that penicillin has a profound therapeutic effect on asymptomatic and early meningovascular syphilis. Less was hoped for from its application to patients with the parenchymatous forms of neurosyphilis. These forms were notorious for their resistance to arsenicals and bismuth and required a combination of fever and specific therapy to be brought to a standstill. It was, therefore, a surprise when gradually reports appeared stating that penicillin exerts a beneficial action on clinical symptoms even in paresis, where it was least expected. However, the workers in this field do not yet agree as to the relative merits of fever and penicillin in general paresis. One group praises penicillin as at least equal to the former methods of treatment with fever; ^{1, 2, 3, 5, 6, 9, 10, 16, 21, 22, 27, 28, 29} the other group still insists that fever is superior to penicillin alone in the treatment of general paresis and advises a combination of both fever and penicillin. ^{7, 8, 12, 17, 18, 19, 23, 25, 26}

To illustrate these differences, I quote first Nicol,¹⁰ who has had unusual experience with malaria therapy for general paresis. He has been affiliated with the Malaria Therapy Center in Horton, England, for many years. He says: "One of the most striking clinical features [of penicillin therapy in general paresis] was the physical and mental improvement in many patients, a phenomenon rarely if ever seen during malaria therapy. Most dramatic results were seen in patients who were confused and in poor physical condition." On the other hand, O'Leary states: "I have never seen a clearcut remission develop in a patient with frank dementia paralytica of any type from penicillin alone, and the partial remissions that occur not only are incomplete but are of short duration. However, penicillin plus fever therapy produce complete remission." An editorial in the 1946 Year Book of Neurology, Psychiatry and Neurosurgery sides with O'Leary and states: "Therapeutic experiences with penicillin in general paresis, including congenital types, and in meningovascular syphilis are very discouraging, and even the remarkable tonic effect has been lacking."

If such diametrically divergent views are given by men who ought to know, how can an outsider form an opinion by reading the literature on this subject? To add to the confusion, all papers on penicillin in neurosyphilis are loaded with statistical fallacies. Since no fast rules exist concerning the optimal amount of penicillin for neurosyphilis in general and for paresis especially, the dosages of penicillin given vary from 2 to 10 or more million units. Some authors combine penicillin with arsenicals, some with malaria, some with both. Some give half the amount of fever which is usual in the fever treatment of general paresis, and so forth. Since the material available for the evaluation of the therapeutic effect is rather small in most of the papers, the statistical computations lack significance. I must admit that from a review of the

literature I would never have been able to determine what the situation really is if I had not had access to my own material at Bellevue Hospital.

How can this confusion of fact and figures best be resolved? I believe that it can easily be done on the basis outlined in the beginning of our paper. To evaluate therapeutic success in parenchymatous neurosyphilis by observing clinical symptoms alone is extraordinarily difficult. This has always been true. The problem arose more than twenty-five years ago when we were called upon to report the results of malaria therapy in paresis. It was then that I began to realize how greatly subjective and emotional attitudes entered into the clinical appraisal of the outcome of malaria therapy. Those authors in favor of malaria treatment obtained a higher percentage of remissions than those opposed to it. Some never saw any improvement from it as long as it was not the accepted form of treatment. It took years before it became a well established fact that malaria therapy of general paresis was the treatment of choice. Careful spinal fluid studies over an extended period of time, however, indicated that malaria was actually an exceptionally effective treatment for neurosyphilis long before this was generally recognized on the basis of its clinical effect. Based on the criteria of activity previously outlined in the description of spinal fluid tests, I was able to forecast with a considerable degree of accuracy whether a patient would remain in remission or would relapse, provided accurate spinal fluid studies were available six months after the completion of treatment. The attempts to evaluate malaria therapy taught me that in some cases it was impossible to reverse physical signs or to restore patients to responsible activity in spite of the fact that the infection was checked. On the other hand, some patients had clinical remissions while their spinal fluid tests indicated continued activity of the infection. To prevent subsequent clinical relapses, these patients had to be retreated.

If we apply the same standards used in the appraisal of malaria therapy for the evaluation of penicillin treatment, we will place greater dependence on the spinal fluid changes following treatment for neurosyphilis than on clinical signs and symptoms. When this is done we find much more agreement among the various investigators of penicillin therapy for neurosyphilis. Every author stresses the fact that increased cell counts first become normal after adequate penicillin treatment and that the total protein values next show a declining tendency. The colloidal gold curves and the titrated complement fixation tests are more resistant but they, too, show a gradual return to lower values. If this is so, and no one denies it, there must be some reason for the favorable tendency in the spinal fluid findings. The conclusion seems inescapable that penicillin exerts a profound therapeutic effect on the pathologic process within the central nervous system and that this effect manifests itself in the spinal fluid findings. Are

we not entitled to assume that the syphilitic process has not been checked but is progressing if the spinal fluid syndrome of some patients shows rising values in all the tests?

What, then, are the results of penicillin therapy for neurosyphilis when measured by the standards of spinal fluid tests? Apart from our own series at Bellevue Hospital there is only one published paper based on sufficiently large material which answers our question and which was followed up for a sufficient length of time. This is the paper presented by Stokes, Steiger, Gammon and co-workers * at the symposium, *Recent Advances in the Investigation of Venereal Diseases*, held in Washington, D. C., on April 15, 1947. The paper describes the effects of penicillin on the spinal fluid in relation to time elapsed after administration. Commenting on the achievement of a normal or "near normal" fluid formula in the various types of neurosyphilis and pointing to some differences in this respect, they state: "These slight differences are, however, less impressive than the massive high favorable proportion of effect and the time in which it is obtained, in aspects of neurosyphilis differing as widely in clinical behaviour as asymptomatic neurosyphilis and paresis."

This statement is in full agreement with our own experience and that of others gained on a smaller number of patients. However, since the clinical syndromes do not parallel the beneficial effects of penicillin therapy on the spinal fluid findings, Stokes and co-workers feel that "these observations suggest that penicillin is operative on the mechanism involved in spinal fluid abnormality, somewhat independently of the mechanism productive of clinical symptoms. All types may expect a spinal fluid improvement. The proportional relationship, which may well be changed by the introduction of another form of treatment such as malaria, having a major effect on symptoms rather than serologic mechanism, has yet to be determined."

This verbatim quotation will illustrate how much confusion exists with regard to the relationship of the pathologic process, the clinical findings and the spinal fluid syndromes. There is still too little understanding that all three are the result of the invasion of the central nervous system by spirochetes. It is the status of the pathologic process, or better, the persistence or disappearance of spirochetes within the central nervous system in which we are interested if we wish to determine the efficacy of a spirocheticidal drug. As we have stated before, following successful treatment there is necessarily no parallel response of the spinal fluid findings and the clinical signs and symptoms, since the latter may be irreversible as a consequence of extensive destruction of nervous tissues. There is also no absolute parallelism between the clinical syndrome and the pathologic process. The fact that there are

* Stokes, J. H. et al.: Three Years of Penicillin Alone in Neurosyphilis. *Am. J. Syph., Gonorr. & Ven. Dis.*, 32:28 (Jan.) 1948.

such entities as asymptomatic neurosyphilis and general paresis in remission proves this unmistakably. But there is, at least in most cases, a constant relationship between the spinal fluid findings and the activity or inactivity of the syphilitic process. As long as this is not fully understood, the reports on the therapeutic effectiveness of penicillin or any other therapy will remain contradictory and, therefore, unsatisfactory.

Results of Penicillin Treatment at Bellevue Hospital.—A few figures are now given on the results of penicillin treatment of neurosyphilis at Bellevue Hospital. All our patients were treated by penicillin alone by intramuscular injections every three hours. The total dosage of penicillin used varied from 2 million to 9 million units, given in individual dosages varying from 20,000 to 40,000 units. The period of treatment varied from nine to twenty-eight days. The longest period of observation was thirty-six months, the shortest six months. Spinal fluid examinations were made before treatment and then every three or six months thereafter. We have so far treated approximately 800 cases of neurosyphilis.* Reliable statistical figures, based on a follow-up of at least six months, are available for only 198 patients. The results reported in Table 1 refer to spinal fluid changes. The clinical improvement in patients treated with penicillin at Bellevue Hospital has compared favorably with that observed following malaria therapy. My own impression of the improvement noted after penicillin is like Nicol's.

TABLE 1

PRESENT STATUS OF 151 PATIENTS (INCLUDING RE TREATMENTS)

	Total	Satisfactory	Indefinite	Failure
Asymptomatic	23	19	3	1
Meningovascular	35	29	4	2
Tabes dorsalis	41	37	3	1
General paresis	33	31	2	0
Taboparesis	19	19	0	0
Total	151	135 (90%)	12 (7%)	4 (3%)

A spinal fluid syndrome which indicated arrest of the process was considered as a satisfactory response to treatment. An indefinite result was assumed when the cell count showed borderline values and the protein content remained high. Those patients whose spinal fluid findings remained unchanged or became worse were classified as failures.

The figures given compare favorably with those for a similar group of patients treated by a combination of malaria fever and chemo-

* This figure has risen to 400. The therapeutic results over a longer period of follow-up are the same.

therapy. A closer scrutiny of this statistical compilation will reveal that the largest percentage of failures occurred in the group of patients with asymptomatic and meningovascular syphilis, because, as a rule, they received smaller amounts of penicillin than those with general paresis. Most of them were re-treated with larger amounts and then responded favorably. It appears, therefore, that the over-all results could be improved by an intensification and prolongation of the penicillin therapy, and that so far there is no need to fall back on methods which are more dangerous and time-consuming.

Finally, in Tables 2 and 3 the actual spinal fluid findings of two cases are presented. They illustrate the dynamic relationship of the spinal fluid syndrome and the tendency of the syphilitic process as it moves towards arrest or further progression. Table 2 gives the spinal fluid findings in a patient whose treatment with malaria was unsuccessful but who achieved an inactive spinal fluid after penicillin. Table 3 illustrates a failure following 2 million units of penicillin and success after retreatment with 8 million units.

TABLE 2
PENICILLIN SUCCESS AFTER MALARIA FAILURE

(E. W., aged 39, white, male. Taboparesis)

Test No.	Date	Blood Wass.	Spinal Fluid Wass.	Colloidal Gold	Total Protein	Pandy	Cells
1	2.13.42	4 †	4 †	55555 †	60	4 †	325/3
February 1942—Tertian Malaria (8 paroxysms) and 10 Daily Mapharsen .06 G.							
2	9.7.42	4 †	4 †	3344 †	35	3 †	21/3
October 1942—Quartan Malaria (9 paroxysms) and 10 Daily Mapharsen .06 G.							
3	2.4.43	4 †	4 †	0111 †	33	3 †	44/3
January–June 1943—20 Melarsen							
4	6.28.43	4 †	4 †	2211 †	35	3 †	5/3
June 1943—January 1944—20 Melarsen							
5	5.29.44	4 †	4 †	0111 †	48	F.T.	18/3
6	10.2.44	4 †	4 †	1111 †	71	4 †	160/3

October 1944—4 Million Units Penicillin

7	10.30.44	12 *	37 *	84 †	56	3 †	54/3
8	12.4.44	12	30	82	45	2 †	1/3
9	2.5.45	9	21	70	43	F.T.	8/3
10	5.22.45	6	20	72	44	F.T.	4/3
11	8.6.45	3	12	43	34	F.T.	3/3
12	1.21.46	4	13	44	31	F.T.	3/3
13	5.13.46	2	6	48	31	F.T.	1/3
14	10.29.46	2	12	52	28	F.T.	3/3
15	4.18.47	3	7	44	30	F.T.	3/3

* Titered in units.

† Reading of first four tubes by the older Lange method of colloidal gold test.

‡ The figure given represents the sum of readings in all 10 tubes by new Lange method.

TABLE 3

RE-TREATMENT AFTER FAILURE WITH 2 MILLION UNITS

(E. H., aged 26, Negro, female. Asymptomatic. Previously treated with 30 injections of Neosphenamine and 34 injections of Mapharsen)

Test No.	Date	Blood Wass.	Spinal Fluid Wass.	Colloidal Gold	Total Proteins	Pandy	Cells
1	4.17.44	4 †	4 †	4444 †	25	†	100/3
2	5.16.44	4 †	4 †	3321 †	25	F.T.	98/3

May 1944—2 Million Units Penicillin

3	5.29.44	100	4 †	1221 †	21	0	15/3
4	7.31.44	84	4 †	1110 †	12	0	2/3
5	11.21.44	66	9 *	50 †	18	0	7/3
6	5.22.45	62	27	107	27	0	70/3
7	6.4.45	62	41	122	25	F.T.	332/3

June 1945—Re-treated 8 Million Units Penicillin

8	7.9.45	53	27	102	24	†	41/3
9	9.11.45	67	19	97	14	0	2/3
10	12.17.45	44	15	58	16	0	1/3
11	3.11.46	41	12	53	13	0	3/3
12	7.2.46	27	10	45	16	0	2/3
13	10.28.46	33	8	55	13	0	1/3
14	4.25.47	28	9	48	18	0	3/3

* Titered in units.

† Readings of first four tubes by the older Lange method of colloidal gold test.

‡ The figures given represent the sum of readings in all 10 tubes by new Lange method.

Briefly summarizing, I am convinced that at present penicillin in adequate dosage is the most convenient and effective treatment for all types of neurosyphilis. Its effectiveness is best evaluated by properly performed tests of the spinal fluid which in most cases reveal whether the syphilitic process is still active, abating or is definitely arrested. The clinical manifestations are important but cannot be used as an authoritative evaluation of the effectiveness of treatment. In most of our cases of neurosyphilis at Bellevue Hospital, the response to penicillin was similar to or perhaps better than that previously observed following malaria.

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CLINICAL ASPECTS OF CEREBRAL LOCALIZATION

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IN our attempts to explain the function of the brain in terms of its structure, various motor, sensory and psychological phenomena have been localized in discrete areas. The evidence for this allocation has been gained from observations in disease in man, and experimental stimulations and ablations in animals. However, improved experimental techniques, the development of procedures for reporting the electrical activity of nervous tissue, and the opportunity for study of ablations and stimulations in man have revised many of our concepts. Neurophysiologists no longer speak of a "center" for a particular function. Actually, no single part of the brain or individual pathway operates exclusively in the execution of any particular function. The brain acts as a whole and in any motor or sensory phenomenon many portions of it are activated. When a finger is moved, in addition to the voluntary movement observed, there is also increased sweating and action of proprioceptors. When the same finger is pricked by a pin impulses travel not only up the lateral spinothalamic tract to the thalamus and the cortex of the parietal lobe, but they also go to the so-called motor cortex, the hypothalamus, the cerebellum, and to other parts of the brain.

Hughlings Jackson conceived of each portion of the brain as being both sensory and motor in function, and this belief is being substantiated by experimental evidence. It has been shown, for instance, that the precentral "motor" cortex receives a large afferent projection from the thalamus as does, of course, the postcentral gyrus. Penfield has stimulated the precentral area in awake patients and produced sensory responses, and removal of the precentral cortex in man leaves a considerable sensory deficit. A single part of the brain also has the property of both exciting and inhibiting neural activity. Thus stimulation of a given point on the "motor" cortex may either initiate a movement or stop one otherwise produced, depending upon the conditions of stimulation.

In health, then, we cannot speak accurately of any one part of the brain carrying out any isolated function. However in clinical neurology it is possible in many instances to correlate syndromes with lesions in specific areas in the brain. In disease or injury a pattern of dysfunction appears, the recognition of which enables us to make a diagnosis of a focal pathological process. This method of localization

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involves certain considerations. The pattern of dysfunction which is produced is not only dependent upon the area of the brain affected but also to a great extent on the rate of development of the causative pathological process. Thus in a slowly developing lesion there may be no neurological deficit for a long period of time because the brain has been able to compensate. The size of the lesion is also very important.

When, in man, the area in the precentral cortex from which arm movements can be obtained on stimulation is removed, the resulting paralysis is brief and leaves little residual defect. However, when both the "arm area" and "leg area" are removed, then the resulting paralysis in both the arm and the leg is much more severe and enduring. Thus, in a sense, the whole is greater than the sum of the parts. It is not infrequently found that lesions in certain areas will give no clinical symptoms. In the monkey, for instance, isolated destruction of the red nucleus or the caudate nucleus gives no observable motor deficit. This does not mean that these structures do not have important motor functions. It does mean that when these parts are destroyed, the brain is able to compensate to a great degree by utilization of other pathways.

It is also important to remember that disease processes actually impair the function of more parts than can be seen on anatomical section. Thus, when one vessel is thrombosed, we may assume that other areas of the brain also have faulty nutrition. Also, by means of the surrounding edema and interference with blood supply, a tumor affects far more territory than it actually directly destroys. The presence of bilaterality of lesions is extremely important. A large amount of tissue destroyed in a cerebral hemisphere does not cause the severe changes that result when even a small amount of tissue is destroyed in both cerebral hemispheres. This is especially true with organic mental symptoms.

The signs of motor dysfunction may, for convenience, be divided into *disturbances of voluntary movement* and *release phenomena*. The latter includes changes in tone, in reflexes, and the production of involuntary movements. All of these may appear in a hemiplegia. The usual pattern of dysfunction in hemiplegia is one which involves mainly the distal musculature of the extremities, dealing with fine skilled movements. The upper extremity is involved more than the lower; there is increased tone, and exaggerated deep reflexes. This is the almost habitual pattern which appears in lesions from the cortex down to the brain stem. A noteworthy exception is the type of paralysis occurring in lesions involving the medial surface and most superior aspect of the hemisphere, such as occurs in parasagittal meningiomas or in occlusion of the anterior cerebral artery. Here the weakness appears predominantly in the lower limb and a foot drop may be produced.

In experimental animals, particularly monkeys and chimpanzees, specific syndromes have been described after the removal of the precentral motor area, a small adjacent anteriorly situated region known

as 4-S or the strip area, and the so-called premotor area, situated more anteriorly. Removal of precentral area 4 is said to produce only paralysis without increase in tone. Removal of the strip gives minimal paralysis but marked increase in tone and reflexes, while forced grasping may be produced when premotor area 6 is ablated. In man, however, such individual patterns are rarely if ever seen, perhaps because disease is not so neatly circumscribed. A hemiplegia resulting from an upper motor neuron lesion has increased tone and reflexes unless there is an accompanying sensory deficit. In a limb in which the proprioceptor pathways are interrupted, flaccidity may be present. In convulsive states produced by focal pathology the physiological differentiation of the various motor areas is useful. Jacksonian seizures produced by more anteriorly placed lesions are more likely to be of the adverse variety in which the head and eyes are turned to the opposite side, the trunk is rotated in that direction, and there are sustained movements of the contralateral limbs. More posteriorly, in the precentral area, the convulsive aura is more sharply focal, commonly involving clonic movements of the opposite thumb and index finger. In frontal lobe and thalamic lesions, signs more commonly associated with disease of the cerebellum may be seen. These include ataxia, dysidiadochokinesis, intention tremor and staggering gait. This is not surprising when it is considered that the frontal area not only sends a large contingent of fibers to the cerebellum but receives projections via the dentato-rubro-thalamic system. Another sign common to both cerebellar and cerebral lesions is the loss of ability to perform discrete movements of an individual finger without moving the others. However, in cerebellar disease the findings when unilateral are usually ipsilateral, while in frontal lobe disease they are contralateral.

For the production of most of the involuntary movements seen in organic neurologic disease, multiple or diffuse lesions are necessary. The *intention tremor* of cerebellar disease can be produced by an isolated destruction of the ipsilateral superior cerebellar peduncle, but for the production of *choreiform* or *athetoid movements*, multiple lesions involving both the cortex and deeper structures are required. Thus the pathological lesions seen in parkinsonism, dystonia, the choreas and Wilson's disease are widespread. A possible exception is hemiballismus, a type of chorea causing wild, violent involuntary movements of large amplitude, occurring mainly in the upper extremity. This has been attributed to destruction of the opposite subthalamic body. A review of the cases in the literature, however, indicates that they occur mainly in older people with vascular disease and with evidence of previous lesions. Cases have also been reported in which the subthalamic body was intact.

Neurophthalmological examination may yield information of value in localization. A discrete cortical lesion rarely yields abnormalities in oculomotor function, which is not surprising in view of the fact that

rapidity of growth of the causative lesion is also a most significant determining factor. In a slowly progressing process, the early mental symptoms are likely to be irritability, anxiety, forgetfulness, diminution of interest, or difficulty in concentrating. In a quickly growing lesion in the same location, the initial picture may be of delirium, disorientation, confusion or stupor.

Mental symptoms may also occur in focal intracranial disease, both above and below the tentorium. In the tumors studied at The Mount Sinai Hospital in the past twenty years, mental symptoms occurred about twice as frequently in supratentorial as in infratentorial growths. In tumors, intracranial hypertension was frequently associated with the presence of mental symptoms. This is not due to the increased pressure per se, but tumors producing elevated pressure are likely to be rapidly growing or close to a ventricle, an apparently favorable milieu for the development of mental changes. For instance, thalamic tumors extending along the wall of the third ventricle have a very high incidence of mental symptoms.

Euphoria and increased psychomotor activity may be seen not only in lesions of the frontal lobes but also in disease elsewhere in the hemisphere and often in lesions about the third ventricle. *Diminished activity and somnolence* may appear in lesions in the same areas. In fact, in the same patient one state may replace another. A *manic type of behavior* with memory loss, hallucinations and confabulation is not rare in lesions about the third ventricle. It may also be produced in tumors elsewhere, particularly those causing subarachnoid hemorrhage. This Korsakoff type of psychotic picture is hardly a focal sign as it occurs in toxic conditions, meningoencephalitis, spontaneous subarachnoid hemorrhage and other diffuse processes. *Hallucinations*, unassociated with other symptoms, do not help in localization. When a formed visual hallucination occurs in association with a defect in the visual field, then it points to a defect in the temporal lobe opposite the field defect. When complex hallucinations with peculiar smells or tastes are experienced, there is again strong evidence for a temporal lobe localization.

In general, mental symptoms must be evaluated along with other neurological manifestations. The more diffuse and bilateral the pathological condition is, and the more rapidly it develops, the more likely one is to see disturbances in behavior.

NUTRITIONAL POLYNEUROPATHY

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AND

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PATHOLOGY

THE characteristic polyneuropathy of nutritional origin involves both the efferent and afferent fibers of the peripheral nerves and is more or less bilaterally symmetrical. Typically, involvement begins in the feet with impaired vibratory sense and position sense, diminution in appreciation of sensation of light touch, pinprick, heat and cold, paresthesias, dysesthesias and hyperactive to absent Achilles tendon reflexes. It may progress up the lower extremities and sooner or later involve both the lower and the upper extremities in a stocking and glove fashion. In extreme cases there may be complete paralysis of the extremities with bilateral foot and wrist drop and the absence of all tendon reflexes. Calf muscle cramps and exquisite calf muscle tenderness are frequent early concomitants of nutritional polyneuropathy and are important diagnostic signs.

Although, typically, nutritional polyneuropathy is bilateral and more or less symmetrical, this is not always the case. Conditioning factors, such as trauma from crutches, impairments in the circulation of the blood, and excessive occupational use, may predispose one extremity, or portion thereof, over any of the others, to damage from nutritional deficiency. Thus a peripheral polyneuropathy seriously involving only the hands may occur on a nutritional basis in a professional pianist, and an individual using a crutch which presses on the axillary nerves may have a neuropathy limited to the arm under which he habitually carries his crutch. Nutritional mononeuropathies may also occur where suitable conditioning factors preexist.

Demyelination of the peripheral nerves has been described in dogs, pigeons and rats maintained on diets deficient in the water-soluble B vitamins and demonstrating clinical neuropathy before death.¹ Similar, but less advanced, lesions have been found in control animals receiving adequate amounts of the water-soluble B vitamins but restricted in total food intake,¹ and it seems likely that inanition may be an etiological factor in the production of peripheral polyneurop-

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athy in both man and animals. Central nervous system lesions characteristic of Wernicke's encephalopathy in man have been found in experimental animals deficient in the water-soluble B vitamins.^{1, 2} In this instance, inanition, per se, has been excluded as a causative agent.

Aring and associates³ obtained biopsies of the terminal portion of the internal branch of the anterior tibial nerve from ten human pellagrins and five normal control subjects. They found that the myelin sheaths were reduced in number in eight of the ten persons with pellagra. The axis-cylinders were found to be involved, usually in the same severity as the myelin sheaths. They also found that mild clinical signs of neuropathy may occur in the presence of an apparently normal myelin sheath content of the nerve. As many of the mild cases of nutritional polyneuropathy of short duration in the human subject respond rapidly to adequate treatment, it is probable that functional changes of sufficient severity to produce clinical signs and symptoms generally precede the development of anatomical lesions.

ETIOLOGY

The fact that polyneuropathy is characteristic of endemic beriberi and the demonstration of the etiologic role of vitamin B₁ deficiency in this disease, led investigators⁴ to study the effectiveness of vitamin B₁ therapy in clinically similar cases of neuropathy not associated with endemic beriberi. Minot, Strauss and Cobb⁴ found the diets of forty-one of forty-three alcohol addicts with polyneuropathy to be inadequate in vitamin B₁ and postulated that this condition in alcohol addicts, so akin to the neuropathy of beriberi clinically and pathologically, resulted from the same cause as beriberi, namely vitamin B₁ deficiency. Subsequently Strauss⁵ and Blankenhorn and Spies⁶ showed that the peripheral polyneuropathy of alcohol addicts undergoes a remission if the patients are treated with diets rich in all the vitamins and supplemented by concentrates of all the B vitamins, even while they are given a pint to a quart of whiskey daily. Jolliffe⁷ and his co-workers carried the work a step further by demonstrating the specific curative effect of crystalline thiamine when used along with a basal diet poor in the B vitamins. As the polyneuropathy of the alcohol addict is apt to be more severe and less responsive to therapy than the nutritional polyneuropathy generally observed in nonalcoholics, it is possible that alcohol may have a serious toxic effect on the peripheral nerves of the malnourished subject. No such effect, however, has been demonstrated in alcohol addicts maintained on diets rich in the B vitamins.

Nutritional polyneuropathy occurs in pregnancy, in the course of chronic, wasting diseases associated with poor dietary practices or disturbances in the absorption and utilization of nutrients, in dietary fad-dists and among persons who, for economic or other reasons, consume diets inadequate in thiamine content. It may occur in persons who

otherwise appear to be well nourished on clinical examination as well as in those obviously malnourished.

The presence of thiamine deficiency may predispose the individual to damage from neurotoxic metals and drugs, but treatment of such neuropathies with dietary measures is ineffective without removal of the toxic agent. Typical nutritional polyneuropathy occurs in diabetics, and responds to nutritional therapy. There is however, in diabetics, a polyneuropathy, indistinguishable clinically from nutritional polyneuropathy, that does not respond to thiamine therapy and cannot be prevented by high thiamine intakes. Apparently, it responds only to control of the diabetes. The origin of this "diabetic neuritis" is unknown.

DIAGNOSIS

The diagnosis of nutritional polyneuropathy is made on the patient's history, his clinical signs and symptoms and his response to therapy. A history of an inadequate diet for a protracted period generally can be elicited. There may be a history of alcohol addiction, or of chronic or repeated infections, or of metabolic disorders, or of pregnancy. The patient's history should be examined for evidences of exposure to neurotoxic substances, for evidences of peripheral vascular disease and for other possible precipitating or conditioning factors.

Nutritional polyneuropathy develops in human beings and animals only after a protracted period of subsistence on a diet deficient in, but not devoid of, thiamine. It is rarely the first manifestation of thiamine or of B complex deficiency. Earlier signs and symptoms include anorexia, weight loss, constipation and diarrhea; depression and irritability, restlessness, fatigability, headaches, memory defects, impairment of judgment and other disturbances in the intellectual and emotional spheres. Both the medical history and the clinical examination should include a search for evidences of such phenomena.

Nutritional deficiency diseases in man are multiple, in most instances, and the clinical examination of the patient with polyneuropathy should include a search for evidences of the existence of deficiencies of nutrients other than thiamine. It is most important to look particularly for signs of niacin and riboflavin deficiency. Deficiencies of niacin, riboflavin and thiamine together largely account for the classical picture of pellagra. Clear-cut evidence of a clinical deficiency in one of these substances presupposes deficiency of the others. The converse, however, is not true.

The earliest evidences of peripheral nerve involvement generally are excessive fatigability of the legs, calf muscle cramps (particularly at night), calf muscle tenderness, burning sensations of the feet and hyperesthesia of the soles of the feet. The tendon reflexes become hyperactive and later may become hypoactive and then absent. Some neurologists choose not to make the diagnosis of polyneuropathy unless



TREATMENT OF HEMIPLEGIA DUE TO CEREBRAL LESIONS

MOSES KESCHNER, M.D.*

HEMIPLEGIA due to cerebral lesions is a sequel to an affection of the motor centers and pathways in the brain. Such an affection may be neoplastic, infectious, degenerative or vascular in nature.

The treatment of hemiplegia resolves itself into, first, treatment of its cause and, later, of the hemiplegia itself. The treatment of its cause may necessitate extirpation of a tumor, evacuation of an abscess, removal of blood clots and foreign bodies (bullets, fragments of bone) in traumatic cases, treatment of an encephalitis, of a blood dyscrasia (purpura, leukemia, scurvy), of neurosyphilis, of cardiovascularrenal disease, or of an apoplexy from hemorrhage or occlusion (thrombosis, embolism) of a cerebral blood vessel.

The most common cause of hemiplegia is apoplexy. In the present state of knowledge there are no specific measures available to prevent apoplexy, yet some of the apoplexies may be delayed indefinitely by the judicious management of patients with arterial hypertension and vascular sclerosis. Patients with cerebral arteriosclerosis, with or without hypertension, are to be cautioned to avoid excesses of any kind. They should be given a light but nutritious diet in small and frequently repeated quantities. In cases with cardiorenal insufficiency there are no indications for a strict lactovegetarian diet; the prolonged use of the latter, sooner or later, produces anorexia, loss of weight and strength, and mental depression. Another cause of unhappiness in these patients is the complete withdrawal of their coffee, alcohol and tobacco. A cup of weak coffee, a cigar after the chief meal of the day, and a glass of beer or sherry before retiring, in those accustomed to such luxuries will, by themselves, not hasten the onset of an apoplexy, and will have a most favorable effect on the patient's morale.

The close relationship between physical and mental processes, and the fact that the latter, especially those referable to the emotions, have a significant influence on the circulation should be given serious consideration before ordering complete mental and physical inactivity for these patients. To be sure, physical and mental overexertion are to be avoided, but this does not mean that the patients must give up their business or occupation entirely and withdraw from all social activities. In this connection it is noteworthy that Gunawardene¹ has pointed out that hypertension rarely occurs among rickshaw runners

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at least abnormalities of the Achilles tendon reflexes are demonstrable, on the grounds that morphological examinations of the peripheral nerves have failed to show any consistent evidence of nerve pathology in cases of lesser involvement than this and because functional disturbances in the legs due to cardiovascular disorders or other conditions producing peripheral anoxia may be the cause of the same symptomatology. The history of the development of the disease in the thiamine deficient subject, however, does not justify any such diagnostic conservatism. From the standpoint of adequate therapy, it is particularly important that the condition be recognized for what it is as early as possible, for the prognosis rapidly becomes worse as the chronicity and severity of the polyneuropathy increase.

The clinical examination should include a detailed examination for lesions of the central nervous system as well as of all the peripheral nerves. Wernicke's disease, a manifestation of severe, and probably relatively acute, thiamine deficiency includes peripheral polyneuropathy, paralysis of the sixth and frequently of the third cranial nerves, ataxia and varying degrees of clouding of consciousness. It has been described in alcohol addicts, pernicious vomiting of pregnancy and in cases of protracted postoperative vomiting. De Wardener and Lennox⁸ described fifty-two cases in a Japanese prisoner-of-war camp; thirty of the patients recovered after treatment with thiamine injections. De Wardener and Lennox emphasize the importance of early diagnosis and treatment. According to them, diagnosis should be based on anorexia, vomiting, nystagmus and emotional changes, before the onset of gross mental and eye changes.

Retrolbulbar neuritis also occurs not infrequently on a nutritional basis with or without an associated peripheral polyneuropathy. Whitbourne⁹ found many cases among school children in Kingston, Jamaica and treated them effectively with a diet of protective foods plus cod liver oil, iron and dried brewers' yeast. She found a definite association between retrolbulbar neuritis and cheilosis and glossitis and in some cases there was concomitant serious impairment of hearing. She reported that those cases "in which the general diet could be improved were cured rapidly, provided an early diagnosis had been made; that is while vision was 6/12 or better. The time taken for restoration of normal vision varies from two months to a year or more depending, it would seem, largely on the general improvement of the diet and of course on the severity of the lesion." Dr. Whitbourne had no opportunity to evaluate the role or effectiveness of the individual vitamins in the production or cure of the neuritis. Strachan (quoted by Whitbourne) described dimness of vision, associated with impaired hearing, in accounts of peripheral polyneuropathy in Jamaica in 1888 and 1897. Roberts and Willcockson¹⁰ described six cases of post-optic atrophy in repatriated prisoners of war, all of whom had severe malnutrition and five of whom were known to have

had beriberi while interned. According to Roberts and Willcockson, during the war there were many reports of visual disturbances in individuals who were forced to live under inadequate dietary conditions. "The incidence of permanent visual impairment was alarming and has been confirmed in many Army and Navy hospitals." Carroll¹¹ found that patients with tobacco-alcohol amblyopia, when treated with crystalline thiamine, while eating diets inadequate "in all known vitamins" and consuming their customary amounts of tobacco and alcohol, made partial or complete recoveries. The most likely explanation of tobacco-alcohol amblyopia is that it is the result of a toxic action of tobacco and alcohol on malnourished cells. Tobacco-alcohol amblyopia probably does not occur in well nourished persons.

TREATMENT

The prime causes of nutritional peripheral polyneuropathy are thiamine deficiency and possibly inanition and therapy should be directed to the restitution of these defects as rapidly and effectively as possible. In addition, all other nutritional defects must be corrected. Nutritional factors, other than thiamine, are quite possibly involved in the production and cure of nutritional amblyopia and deafness. In general, also, the individual vitamins appear to be most effective when the diet is made as adequate as possible.

The diet should contain sufficient calories to enable the patient to achieve or maintain his optimum weight. It should contain at least 150 gm. of protein for an adult and proportionate amounts for children. It should at least meet the recommended allowances of the Food and Nutrition Board¹² in respect to the minerals and vitamins. One tablespoonful of dried brewers' yeast three times daily is beneficial, when tolerated. The routine daily use of an oral polyvitamin preparation containing vitamin A, 25,000 I.U.; thiamine, 5 mg.; riboflavin, 5 mg.; niacin, 50 mg., and ascorbic acid, 100 mg., is recommended for at least the first two or three months of therapy. An additional 10 to 15 mg. of thiamine should be given daily, in two to three divided doses, for at least the first month of therapy.

Parenteral vitamin therapy is not indicated unless the patient cannot be fed by mouth or it is suspected that the deficiency is secondary to difficulties in absorption from the intestinal tract. In severe Wernicke's disease it is probably desirable to give daily, in divided doses, from 25 to 30 mg. of thiamine intramuscularly or intravenously until consciousness is restored. Also, in niacin deficiency encephalopathy (not discussed in this paper), 500 mg. of sodium nicotinate should be given parenterally daily until recovery. Lesser degrees of obvious niacin deficiency can be effectively treated with a total daily dose of 300 to 500 mg. of niacin by mouth. The treatment of obvious riboflavin deficiency requires a daily dose of 10 to 20 mg. of riboflavin by mouth.

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in India who take violent exercise over long and exhausting hours, whereas it is notably common in certain social groups who habitually lead sedentary lives with overeating. Patients with arterial hypertension should not be constantly reminded of their high blood pressure, nor are they to be threatened with the dangers of a "stroke," lest they exclude themselves from all activities and pleasures of life. Such injudicious management of patients with vascular disease not infrequently produces a fear and anxiety neurosis, if not an actual depression which may be more menacing to their psychosomatic integrity than the onset of an apoplexy for the alleged prevention of which the treatment was originally instituted.

TREATMENT OF APOPLEXY

Essentially the treatment of apoplexy due to hemorrhage differs very little from that due to vascular occlusion.

In most cases the diagnosis of the cause of apoplexy can be made only with a certain degree of probability; the degree may be so high as to almost reach certainty, or, on the other hand, so low as to amount to practically little more than a guess; unfortunately in some cases the diagnosis has to remain doubtful for a long period.

If it is not definitely certain whether the lesion is hemorrhagic or thrombotic the following measures are advisable. The patient is to be kept in a cool, darkened room in the recumbent position with the head and shoulders slightly elevated and tilted to one side to avoid obstruction from the tongue; the clothes about the neck should be loosened and flexion of the neck avoided. If the patient is restless, sedation is indicated in the form of paraldehyde 8 to 15 gm. (2 to 4 drams), preferably by rectum, or with barbiturates (intravenously); morphine should be avoided. If the patient is unconscious or semiconscious, nourishment is to be given by gavage, or by venoclysis. If the patient is conscious nourishment is given by mouth, care being taken to prevent coughing; at first, fluids, supplemented with vitamin and fruit juices, and later, a milk diet with a gradual return to an ordinary diet is indicated.

Headache, in cases with increased intracranial pressure, is best relieved by lumbar puncture, not more than a few cubic centimeters of spinal fluid being withdrawn in the first instance, lest the sudden lowering of pressure cause a shift of the cranial contents with the danger of a recurrence of the bleeding. If necessary the puncture may be repeated. Another useful method of reducing intracranial hypertension is the intravenous injection of from 50 to 100 cc. of 50 per cent glucose or sucrose; too often, however, this causes dehydration of the tissues, so that it may be more advisable to avoid hypertonic solutions and to inject 5 per cent glucose in saline. Applications of ice bags to the head to relieve headache are of little or no value.

In cases of cerebral hemorrhage some physicians resort to venesection.

tion; I have never been convinced of the therapeutic effect of venesection after the bleeding has once begun. If used at all, its indications are a strongly acting heart with definite ventricular hypertrophy, full and incompressible peripheral arteries, pulsating carotids and a turgid face; in such cases 10 to 12 ounces of blood may be withdrawn.

In diabetics with hemiplegia receiving insulin there may occur periodic aggravations of the paralysis, or a so-called insulin paralysis may set in from an acute hypoglycemia; in these patients it is imperative to maintain a proper insulin balance.

In patients with hemorrhage from a branch of the middle cerebral artery, compression of the carotid on the side of the bleeding vessel is advocated as a measure to be employed to arrest the bleeding; I have employed this method but was not impressed with its effectiveness. If the procedure is resorted to, one must be absolutely certain of the diagnosis of hemorrhage, because if a thrombosis is the cause of the apoplexy, the method would favor an extension of the clot.

In cases of severe cerebral hemorrhage a trephine followed by evacuation of the blood clot is indicated. Operation is justifiable in young or middle-aged individuals who have survived twenty-four to forty-eight hours after the initial bleeding and show evidences of progression of symptoms. In some cases a preliminary ventriculography for precise localization of the lesion is advisable. Patients with advanced arterial hypertension and renal disease, or with massive cerebral hemorrhage as revealed in the spinal fluid, are not suitable for operation.

In cases of cerebral bleeding following injury to the brain, exploration in the form of a large decompression on the side opposite the paralysis is indicated; the decompression may relieve partially the cerebral edema, even though the intracranial bleeding be diffuse.

In cases of diffuse hemorrhage in the brain due to the blood dyscrasias, the subcutaneous injection of 25 to 50 rat units of theelin may arrest the bleeding. Repeated small (250 cc.) blood transfusions may also have a similar effect.

In cases of intracranial hemorrhage in purpura haemorrhagica in which the onset of the bleeding is not apoplectiform, and the symptoms less alarming, transfusion and splenectomy may be considered; it must, however, be borne in mind that splenectomy is a serious operation, and in most types of secondary purpura as well as in the cases of idiopathic hemorrhagic purpura it does not control the bleeding.

INTRACRANIAL HEMORRHAGE IN THE NEWBORN

One of the principal causes of this form of intracranial bleeding is difficult parturition. Prophylactic treatment in the nature of good midwifery offers perhaps the best hopes in its prevention. Once the hemorrhage has occurred very little can be accomplished unless, in desperate cases and when the bleeding can be localized, trephining

and evacuation of the clot is attempted. The hazard of such an operative procedure in newborn and young infants is so great that most neurosurgeons are skeptical about the advisability of operating. Infratentorial bleeding is certainly not amenable to operation, and in supratentorial bleeding even if the blood clots can be successfully removed, the damage to the underlying brain tissue from compression by the clot renders the prognosis for a complete recovery extremely doubtful. In cases of supratentorial bleeding with a bulging fontanelle and evidences of marked increase of intracranial pressure, lumbar puncture may afford temporary relief.

In cases in which the intracranial bleeding is due to hypoprothrombinemia, vitamin K and its esters is a specific remedy; the bleeding usually ceases promptly following an intramuscular injection of 0.5 mg. of the vitamin K analogue (sodium-2-methyl 1, 4-2 naphthhydroquinone disulfate).²

SUBARACHNOID HEMORRHAGE

Cases of subarachnoid bleeding are by far the most amenable to treatment. The general treatment of the attack itself does not differ from that of intracerebral hemorrhage but, in addition to the general measures employed, the removal of 15 cc. of spinal fluid by lumbar puncture is generally followed by a prompt amelioration of most of the distressing symptoms. Should the symptoms recur or persist, repeated withdrawal of the bloody fluid is indicated.

With the persistence of the bleeding and the appearance of focal signs, even though repeated lumbar punctures have been employed, it is advisable to subject the patient to ventriculography for the possible visualization of the site of the bleeding which may be in the nature of an aneurysm or some other vascular anomaly amenable to surgery. In the diagnosis of intracranial aneurysms, angiomas, arteriovenous aneurysms and similar vascular abnormalities which may give rise to repeated intracranial bleedings, intracranial angiography^{3,4} yields much more reliable information than intracranial air studies.

SUBDURAL HEMATOMA

An intracranial hematoma large enough to produce focal signs and symptoms of cerebral compression should be evacuated as soon as the diagnosis is established, regardless of the presence or absence of paralysis.

CEREBRAL THROMBOSIS

In the treatment of cerebral thrombosis, in contrast to that of hemorrhage, the aim is to diminish the tendency to clotting. Cardiac action, therefore, should be strengthened, if necessary, by the administration of cardiac stimulants. For patients with cardiac failure caffeine is probably the most useful drug; it is best administered hypodermically in the form of a sterile solution of caffeine with sodium benzoate,

an average single dose being between 0.3 and 1 gm.; frequency of repetition depends on its effects; its action usually lasts about two hours; it is an emergency remedy and, when no longer needed, should be discontinued.

If the patient complains of severe headache, moderate doses of the coal tar products may be given. If no relief is obtained lumbar puncture is indicated. Venesection is absolutely contraindicated. If restlessness is a marked feature, small doses of the bromides, the barbiturates, paraldehyde and even codeine may be given.

In cases of cerebral thrombosis due to cardiovascular syphilis, the usual antisyphilitic remedies should be administered. In this connection it is well to bear in mind that whatever damage to the cardiovascular system has been produced by the syphilis cannot be repaired so that the only hope of treatment is that it may prevent the occurrence of fresh lesions.

Cases of polycythemia are not infrequently complicated by cerebral thrombosis. The treatment of the thrombosis in these cases does not differ from that of thrombosis from other causes.

THROMBOSIS OF CEREBRAL SINUSES AND VEINS

In primary thrombosis of the cerebral sinuses and veins, which usually occurs in patients with general malnutrition and prostration, very little can be accomplished by treatment other than maintaining the nutrition and circulation, the former by overfeeding and a high caloric diet, and the latter by cardiac stimulation and blood transfusions. When there is evidence of the extension of the thrombosis, the administration of heparin should be considered as soon as the condition is recognized.

The treatment of secondary thrombosis is in most instances surgical; drainage of retained discharges should be established. In the septicemic forms, bacteriophages, penicillin, the sulfa drugs and transfusions should be employed. In sinus thrombosis secondary to disease of the ear or of the accessory sinuses prompt surgery is indicated; in such cases the jugular vein on the affected side is to be ligated following the opening of the sinus and liberation of the thrombus.

CEREBRAL EMBOLISM

The treatment of cerebral embolism is essentially the same as that of thrombosis; in embolism the heart is more apt to be irregular and, if its action is also feeble, vegetations may become detached from the valves with greater facility; in view of this, attention to cardiac action is most important.

AFTER-TREATMENT OF APOPLEXY

Confinement to bed following an attack of apoplexy varies with its severity; two weeks in mild cases, and four to six weeks in more severe cases.

In the after-treatment—and this applies to thrombosis as well as to hemorrhage—massage and passive motion is essential to minimize the spasticity, to prevent ankylosis of the joints, and to maintain the trophic condition of the affected parts. Gentle massage, in the form of effleurage (light stroking movements) and passive motion is advisable even early and while the patient is still recovering from the acute effects of the apoplexy. Later, more vigorous massage and reeducation of the muscles to reestablish the pattern of motor functioning with a perspective for proper psychomotor orientation is to be resorted to. The patients must be taught how to walk and take care of their daily needs. It is remarkable to see how the morale of these patients rises when they are taught to walk with a crutch or cane, to lace their shoes, to cut their meat, and to engage in similar activities.

For the posthemiplegic athetoid movements in which incoordination is a prominent feature, gymnastic exercises in the form of rhythmic movements of the fingers and thumb are indicated. In cases of unilateral athetosis following birth injury, extirpation of the opposite arm area in Brodmann's area 6 is followed by a diminution of the athetoid movements.

If flexion contractures develop, the application of a pad soaked in magnesium sulfate over the affected flexor muscles and connected with the positive pole of the galvanic battery (magnesium iontophoresis) not infrequently causes relaxation of the spastic flexors; the beneficial effect of this procedure is thought to be due to a curare-like influence exerted on the myoneural junction. Muscle spasm in hemiplegia of cerebral origin has been reported to be relieved by the subcutaneous administration of neostigmine methylsulfate, the usual dose being 1 to 2 cc. of a 1:2000 solution with a suitable amount of atropine to prevent the untoward effects of the neostigmine; the injections are usually given three or four times a week. Similar results are claimed from the use of tridione, given orally in doses from 0.3 to 0.6 gm. three times a day. I have employed neostigmine as well as tridione, but have observed no demonstrable beneficial effects when the contractures were solely due to hypertonicity from involvement of the pyramidal or of the extrapyramidal motor systems.

In some cases of infantile hemiplegia with motor handicap due to permanent contractures, single or multiple tenotomies may have to be performed. In cases of so-called "scissor leg," the hypertonicity of some of the muscles has been successfully overcome by dividing the nerves to the adductors of the thigh, or by Foerster's operation of cutting the sensory roots in the lumbar segments of the spinal cord.

In cases of localized epileptiform seizures following hemiplegia, especially in posttraumatic cases, electroencephalographic and intracranial air studies may reveal scars, meningocortical adhesions or encysted fluid which act as epileptogenous zones. The surgical removal of such foci is followed, in a number of instances, by a diminution in

the frequency and severity, and in some instances, by a total disappearance of the seizures. Phenobarbital and dilantin, in appropriate doses, are depended on for the control of grand mal, psychomotor and focal types of seizures. The addition of tridione (in doses of 0.3 to 2.1 gm. a day, depending on the age and weight of the patient) to the phenobarbital and dilantin, aids in the control of petit mal and/or psychomotor seizures which may coexist with the grand mal attacks.

TREATMENT OF APHASIA

Educational therapy is to be employed in cases of aphasia complicating hemiplegia. A slight aphasia usually disappears spontaneously. In the more severe cases with serious damage to the motor and sensory speech mechanisms in the left side of the brain, in right-handed individuals, there is employed a process of training designed to activate the right side of the brain, so as to mobilize the right-sided speech mechanism. In some cases this procedure may be successful early; in others it may never be followed by a return of voluntary speech. In the former, systematic training of the left hand in the performance of voluntary acts that had previously been performed only with the right hand is indicated. In cases of word-deafness (inability to understand spoken words) simple directions are at first given to the patient, and later, more complicated ones; if the patient experiences difficulty in reviving word images he must be trained in naming objects. In motor aphasia (inability to speak spontaneously) the patient should be made to repeat words after another person. Similar exercises are to be employed in reading and writing; in patients with motor aphasia attempts to write are useless until there has been some return of articulate speech. It is best to begin with simple exercises, followed gradually by more complicated exercises. The exercises should never be continued long enough to fatigue the patient; it is more advisable to give them several times a day for short periods. Patience and perseverance with encouragement and reassurance are necessary to obtain favorable results.

Finally, the after-treatment of patients with hemiplegia comprises regulating the patient's life so as to avoid physical stresses, anxiety, worry and emotional upsets. As soon as there is evidence of even partial restoration of function, a program of activities must be outlined that will be most effective in hastening the patient's readjustment to an industrial, social and emotional life compatible with his condition, so that he may regard himself as still being a useful member of society, lest he lead a merely vegetative life and become mentally and physically deteriorated.

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MULTIPLE SCLEROSIS

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MULTIPLE sclerosis is the commonest of the diseases in which the chief lesion is of the myelin sheaths. The symptoms and the signs are numerous, diffuse and spotty in distribution because of the fact that the myelin in various portions of the nervous system is involved. Descriptions of the disease were given in 1835 by Cruvelhier and in 1837 by Carswell, a London Medical student. Wide understanding of it dates from the papers of Charcot in the 1860's and 1870's.

Incidence.—First and most tragically, multiple sclerosis is chiefly a disease of young people though no adult is wholly safe from it. It occurs frequently in the second decade, begins very commonly in the third, often in the fourth, sparsely in the fifth and sporadically thereafter.

Multiple sclerosis occurs somewhat more often in females than in males, and it has an interesting geographical distribution, being rare in the tropics and also in China (Snapper). At least some areas in our own extreme South are reported to have little multiple sclerosis. Steiner was able to find only one case in New Orleans, in a recent year. The incidence in the United States would seem to be about 3 per cent. Figures collected by Wechsler¹ show an incidence among all neurological cases in the United States of 365 up to 1914; from 1914 to 1921 the American incidence (chiefly in New York City) was 1.1 per cent. The incidence among Europeans was 1.33 per cent.

Curiously enough, several observers have noted considerable concentrations of patients with this condition in certain small towns. A new, broad statistical study is under way now, under the auspices of the National Multiple Sclerosis Association. The National Multiple Sclerosis Association is a new organization whose purpose is to centralize knowledge of the disease and of investigations into its cause, and to promote research.

CLINICAL FEATURES

Clinical Types.—Marburg has defined four clinical types of multiple sclerosis. The commonest and most characteristic type is (1) that with *outbursts* (attacks) and *remissions*, and (2) the chronic *progressive* type ranks next in frequency, followed by (3) the *stationary* type, and (4) the acute type (acuteness of onset of a massive array of dis-

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turbances). These four categories are useful although they are not entirely distinct from each other; for instance, acuteness of onset has no relation to the future course in terms of type 1 or type 2. Quite commonly, a case which for a long time has gone on with outbursts and remissions ultimately becomes chronically and steadily, although slowly, progressive. Moreover, suddenness of onset is highly characteristic for individual small lesions; the *total* onset may appear gradual, as some days may pass before all of the lesions have occurred; the difference between this common mode of onset and that in which *all* the lesions develop within a few hours, is perhaps only a matter of degree.

The same sites are likely to be involved in each new attack; for example, if in the first attack there are numbness of the right hand and rigidity of the left leg, the same phenomena, with the addition of new ones, are apt to recur in later outbursts. As the years pass, remissions are likely to be less and less complete because the lesions in the central nervous system have become more and more fully replaced by glial scars in each attack. Finally remissions cease to occur, and the patient's condition becomes either stationary, or chronically progressive.

An important question is whether the chronic progression may not really be an aggregation of imperceptible minute attacks.

Early Symptomatology.—The common early symptoms are these: disturbances in the ocular muscles, which are partially paralyzed so that vision is blurred and doubled; sensory disturbances, which in the beginning are likely to be chiefly subjective—numbness and tingling are the commonest of these—and which may appear anywhere in the body, but particularly in the hands and feet. Occasionally a patient will notice first that he does not feel the shaving process normally. The sensory symptoms are often described as the feeling of a girdle around part of the chest or abdomen. Involvement of the pyramidal pathways may occur early, with resulting weakness, paralysis and spasticity, chiefly of the legs but by no means excluding the arms. The cerebellum and its pathways are likewise frequently affected early, resulting in ataxia of the extremities and also of the trunk. This combination may lead to the so-called "washstand syndrome," in which the hand-washing patient finds himself shaking in front of the washbowl and has to lean on it to keep from falling into it. Another common early symptom refers to the bladder; all varieties of bladder dysfunction may occur in the disease, but the commonest are urgency, frequency and incontinence. Rarely is there rectal incontinence; constipation is common.

A first attack consists of the appearance of one or several, and occasionally many, of these phenomena. They may occur in any order. Any one may occur suddenly in completed form, although several days are required for completion. The array which is going to con-

stitute completeness for the individual attack may take a week or two to develop, even though each individual lesion may occur suddenly, so that, during the evolution of the attack, the picture may show many changes.

The disease may then become stationary or the patient may slowly regress in the so-called chronic progressive fashion, but most commonly the patient begins to improve, going into what is called spontaneous remission. It is probably correct to say that when he is in a remission, the patient does not *have* multiple sclerosis, the signs and symptoms seen during the remission being residua of the disease, like the valvular scars which follow acute rheumatic fever.

The Signal Symptom.—A special, sometimes important point in the clinical picture is the so-called signal symptom. Often a patient with multiple sclerosis, if pressed, may be able to recall a fleeting symptom years before, such as numbness of the fingers which disturbed his writing for perhaps an afternoon. Little attention is paid to such events at the time of occurrence, and they are not likely to be seen by doctors. Conceivably they occur far more often than we think. It is not impossible that the minor events of this kind that have been experienced by most of us, really represent multiple sclerosis which is never heard from again.

Late Symptomatology.—As the disease progresses, either gradually or in attacks, other developments are usually superadded. Although the phenomena already described are likely to dominate the early scene, any symptoms may occur at any time.

Optic Nerve Disturbances (Retrobulbar Neuritis).—Persistent blindness occurs very rarely but vision may be permanently markedly diminished, most often in the form of small scotomas. Rarely gross visual field defects such as hemianopsia occur. If the scotoma is a central one vision is correspondingly greatly reduced. The late Dr. Robert Lambert, until recently attending ophthalmologist of the Mount Sinai Hospital, discovered the important fact that scotomas, if carefully plotted, frequently show themselves as a collection of small defects rather than as one massive blind area. Thus the disseminated, scattered nature of the disease may reveal itself in the minute study of what casually appear to be single phenomena. The disease is disseminated both in time and space.

Ocular Muscles.—Paralysis of the lateral rectus muscle, producing diplopia, is common; other muscles are weakened or paralyzed more rarely. However, lesions in the posterior longitudinal fasciculus were also frequently seen, with resultant dissociations of movement of the two eyes. This can be even more disturbing to patients than double vision. Oscillopsia (oscillation of vision during walking, when treading jars the body) also occurs occasionally. This is probably a sign of cerebellar dysfunction. Nystagmus, also a cerebellar phenomenon, is a common manifestation.

equilibrium. Even *lumbar puncture* should be avoided if possible, since regressions sometimes follow it.

Emotional disturbances are equally important as precipitators. Precipitation, however, should not be confused with cause. Superficial thinking sometimes leads doctors to assume that because a certain factor, especially emotional stress, can induce attacks in recurrent disorders, these factors are the basic cause of the disease.

Fatigue.—An attack may be initiated by any kind of fatigue: walking too hard or too far, or receiving too much massage, for instance, may induce an attack. Not infrequently patients have attacks after long automobile drives. Every kind of fatigue—mental, emotional or physical—is dangerous for people with multiple sclerosis.

Nutritional Inadequacy.—Some patients with multiple sclerosis have a notable aversion to dairy or other fats; some others avoid vegetables. No *specific* relation between chronic dietary inadequacy and the incidence of multiple sclerosis has been shown. However, patients with proper nutrition tend to do better than others over a period of years, a fact independently observed by Brickner and Brill⁴ and by Putnam⁵ who found that private patients had a generally more favorable course than those at the Boston City Hospital Clinic. In addition, obese patients appear to regress more slowly than thin ones.

Marked adverse changes in diet sometimes precipitate new attacks. Reducing diets should be used only with great caution.

PROGNOSIS

Unhappy though the prognosis is, it is not as bad as painted in many textbooks. It is common for patients to continue in a state of reasonable function for as long as eight or ten years, and much longer periods are not rare. One patient of my acquaintance walked and functioned fairly well for thirty-one years, being able even to walk on ice in winter with the aid of a cane in the sixteenth year of the disease. To be sure, most patients do progress ultimately to an almost helpless condition, but the progress is very slow indeed. The usual causes of ultimate death are infections of decubitus ulcers or of the urinary tract.

PATHOLOGY

The primary site of disturbance is the myelin sheath in various scattered areas in the central nervous system. The sheath disintegrates and the debris is absorbed in large phagocytes. After the process is under way the glia begins to proliferate until glial scars are formed. Later, the lesion becomes entirely scar. These hard, scattered scars, actually end products, are what led Charcot to name the disease *sclérose en plaque*. Later, either due to extension of the process or perhaps to pinching of the scar, the axone itself becomes affected and dies. When that happens all chance of recovery of the particular lesion involved is ended.

During the stages in which axones are merely demyelinated, in patches, the nerve impulse evidently can still be transmitted.

What happens during a remission is totally unknown. Although we believe that myelin in the central nervous system cannot be redeposited once it has vanished, yet during the remission something happens to restore the nervous system to normal or almost normal function. It is conceivable that if the myelin is only injured and not totally destroyed, it may be able to reconstitute itself. Another conceivable type of remission is described below.

DIFFERENTIAL DIAGNOSIS.

Spinal Cord Tumor.—One condition which occasionally must be differentiated from multiple sclerosis is spinal cord tumor. Every now and then in multiple sclerosis, a lesion cuts nearly all the way across the cord. Clinically such cases may simulate tumor, particularly because the symptoms and signs indicate a fixed level for the lesion, the evidence of dissemination being obscured. Spinal puncture and myelography usually settle the differential diagnostic problem, but not invariably. When level lesions of this sort appear suddenly, they require immediate operation if they are tumors, so as to reduce the risk of permanent damage to the spinal cord. If other evidences of multiple sclerosis are doubtful, as in one such patient who had an additional story of previous questionable slight numbness in one hand, and myelography is inconclusive, surgery may have to be invoked. Myelography may even be positive in multiple sclerosis; a patient operated on by Dr. Bronson Ray proved to have localized edema of the cord, sufficiently massive to cause a block by myelography, resulting from an acute attack of multiple sclerosis. Laminectomy seems to do no harm in multiple sclerosis, a fact brought out years ago by Dr. Charles Elsberg.

Syphilis.—In this disease (not tabes) the spinal cord is apt to reveal mainly the picture of bilateral pyramidal tract injury which resembles Erb's spastic paralysis. Of course, the spinal fluid serological study establishes the diagnosis. Some French authors, in particular, have spoken of multiple sclerosis as caused by syphilis, but this is probably no more than an association since there is nothing mutually exclusive about either of these diseases.

Erb's Spastic Paralysis.—This is an involvement of both pyramidal tracts for unknown reasons, with neither the presence of syphilis nor the story that belongs to multiple sclerosis. Occasionally the full picture of multiple sclerosis develops later.

Amyotrophic Lateral Sclerosis.—This disease characteristically involves the pyramidal tract and the anterior horn cells; it is the one condition in which a combination of upper motor and lower motor neuron involvement is habitually seen. Flaccid paralysis, atrophy, fibrillations, and the electrical reaction of degeneration occur in conjunction with

increased reflexes. Since the lower motor neuron is not completely destroyed, the hyperreflexia due to the pyramidal injury is apparent. This disease terminates fatally much more quickly as a rule than does multiple sclerosis although occasionally patients with it are seen who have survived a long time. One recent patient presented the typical picture of amyotrophic lateral sclerosis, but curiously on the left side only; the affliction had endured for more than thirty years. In amyotrophic lateral sclerosis, the bulbar structures are more apt to be involved than they are in multiple sclerosis; the conspicuous lower motor neuron implication, the unremitting course and sometimes the marked bulbar involvement in amyotrophic lateral sclerosis are inescapable diagnostic points.

Encephalomyelitis.—Some believe this condition to be a virus infection, and others, led by Putnam, think it identical with multiple sclerosis. The lesions are disseminated and may appear anywhere in the central nervous system. The picture of multiple sclerosis may easily be simulated. If fever is marked (low fever can occur in attacks of multiple sclerosis) or if the spinal fluid contains a great many cells (50 cells would be an extremely high count for multiple sclerosis), the indications obviously point to an infection. The patient may recover only to have a recurrent attack later. Some cases, at first diagnosed encephalomyelitis, ultimately become frank cases of multiple sclerosis.

The differential diagnosis may be impossible to make. The points mentioned above (cell count, fairly high fever) and occasional pictures completely atypical for multiple sclerosis, are the best criteria.

Neuromyelitis Optica (*Devic's Disease*).—In this condition optic nerve atrophy and pyramidal tract involvement predominate. It is apt to run a very acute course, and to be very severe. Probably the disease is a fulminating form of multiple sclerosis. Pathologically, there are infarcts but in the view of some authorities the infarcts do not distinguish the condition from multiple sclerosis.

Dorsolateral Sclerosis Due to Primary Anemia.—The involvement is almost uniformly limited to dorsal columns and the pyramidal tract. This can occur, and often does, in multiple sclerosis. Differential diagnosis is established by the blood findings. A point to keep in mind in this connection is Dattner's report that in multiple sclerosis the hydrochloric acid content of the stomach may also be reduced or absent.

Hysteria.—One important occasional necessity is the differentiation of multiple sclerosis from hysteria. Particularly in the early phases of some cases of multiple sclerosis, when the symptoms are sometimes entirely sensory, the diagnosis of hysteria is occasionally erroneously made. This is particularly true if the patient happens to be a hysterical person. Considerable neurological experience may be necessary to permit a proper evaluation of the patient's description of his numbness or tingling. However, if any neurological sign can be found, hysteria becomes merely a factor—possibly a precipitating factor to be

sure. Multiple sclerosis is to be thought of in any young person who rather quickly develops neurological phenomena, especially if they are in the categories above-mentioned.

ETIOLOGY

The prospects for discovering the etiology appear more promising now than ever before.

Some years ago, Putnam announced the finding of thrombi in venules associated with lesions. He subsequently showed that blockage of the venous overflow of cortical areas produced more degeneration of myelin than of axis cylinders. He is responsible for the theory that lesions are caused by thrombi in venules.

Other studies were also centered around the circulation.* The indiscriminately scattered distribution of the lesions suggested that something emanating from the blood stream might be responsible for the lesions. Abnormal lipolytic activity was sought and some evidences of it were found, but in all likelihood these are not of primary importance. Marburg had independently suggested that an abnormal lecithinase might be responsible.

In 1934, Rivers and Schentker⁷ discovered that monkeys, receiving a long series of injections of emulsified rabbit brain, sometimes developed a condition resembling human multiple sclerosis. The lesions in monkeys appear much more intensely inflammatory than those of multiple sclerosis. The difference may be a species difference, or possibly due to the acuteness of the process in monkeys or it may be that the disease is not identical with multiple sclerosis. The phenomenon is, of course, reminiscent of what sometimes happens in rabies treatment; occasionally a patient undergoing that treatment develops a condition resembling multiple sclerosis. Recently, Freund has devised a mixture which greatly speeds up reactions like that observed by Rivers and Schwentker. This finding was immediately taken up by Kabat,⁸ by Morgan⁹ and by Morrison, who have been able to reproduce the Rivers' phenomenon with one or two injections into monkeys and have consequently given a tremendous impetus to the earlier studies. Brain substance from fetal rabbit's brain (without myelin) does not produce the result. Two or three of Kabot's monkeys have been reported to have had remissions and recurrences of their visual symptoms.

An attempt to desensitize patients to brain substance was prompted by the work of Rivers and Schwentker some years ago. Most of the patients promptly became worse. One regressed rapidly to death.

Another approach resulted from the observation of Franklin¹¹ that the retina of a patient who suddenly lost left eye vision in Franklin's presence was blanched. The vessels reopened and vision returned in a few minutes. The patient had an undiagnosed, disseminated neurological disease, possibly multiple sclerosis. Later a patient was observed by the author, with true multiple sclerosis, and with the complaint of blurred vision following hot baths. Retinal arteriolar constrictions

spasticity and general improvement in the use of muscles as a result of reeducation may be hoped for. Prostigmine or curare may be of added help in the treatment of spasticity.¹⁴ H. Kabat's employment of the automatic movements sometimes associated with massive spinal cord implication, in the interests of useful movement, is of considerable interest.¹⁵

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THE ADVANTAGES OF THE METHOD OF SIMULTANEOUS STIMULATION IN THE NEUROLOGICAL EXAMINATION

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THE customary method of examining sensory function is to stimulate a single point and to note the subject's response to the stimulus. Generally speaking this form of testing is adequate in our study of disorders of sensation. There are cases, however, in which single stimulation fails to reveal any defect yet double simultaneous stimulation will show a marked loss of sensibility at one of the points tested.*

* By double simultaneous stimulation we mean that two areas, one on each side of the body, are stimulated simultaneously and the patient is required to report what he perceives. Simultaneous testing may also be carried out on the same side of the body but the distance between the two points is much greater than that used in the ordinary "compass test." In the "compass test," which measures two point discrimination, the distance between the two points is relatively short.

The method of double simultaneous stimulation was described by Oppenheim in 1885. He reported three cases of loss of sensation which could be demonstrated only upon double simultaneous stimulation. In his textbook, he refers to this method as follows. "In certain brain diseases which cause unilateral disturbances of sensibility the following procedure is advantageous. Stimulate simultaneously two symmetrical points. The patient will always detect it only on the sound side. This manner of examination we would call the method of double stimulation." Since then there have been sporadic reports of cases in which this method of double testing was found to be advantageous.

During the past four years we have used the method of double stimulation routinely and found in this way many more cases of impairment of sensation than we could have found by ordinary tests with single stimulations. Defects in cutaneous, visual and auditory perception were thus demonstrated.* The most distinct changes in sensation were apparent in tests of the visual or cutaneous modalities. This was especially true, when the stimuli were applied on either side of the median plane of the sensory field tested; in visual tests the stimuli were applied simultaneously on either side of the fixation point, while in examination of the cutaneous modalities they were applied at corresponding points on the right and left side of the body.

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ILLUSTRATIVE CASES

The following cases illustrate the advantage of the double over the single method of stimulation.

CASE I.—A 29 year old Marine with a gunshot wound through the left parieto-occipital lobes complained that his vision blurred on the right side whenever he looked at an object for more than a few seconds. Letters on a printed page appeared to be clear but on continued reading they faded on his right side. When he blinked, his vision became normal. Routine perimetry with single test objects disclosed normal fields of vision for perception of movement, form and color. However, with the method of bilateral simultaneous stimulation the patient showed a right homonymous hemianopia. When two objects were exposed simultaneously, one on each side of the fixation point, our patient saw the one in his left half field of vision; the object on the right appeared dull or became totally invisible or "extinct." * As soon as the object in the normal (left) half field was withdrawn the patient clearly perceived the object remaining in the affected (right) half field of vision. Moreover it was found that the more visual stimulation there was on his left side, the less he perceived on the right side of the fixation point. The visual defect on the right was thus apparent only when both sides were simultaneously tested.

Comment.—In this case there was no question of localization. It was known that the patient was injured in the left parieto-occipital region. The problem was to find the basis for his visual complaints. Standard perimetry offered no help. It was only after the elicitation of a right homonymous defect on double simultaneous stimulation that we were able to explain blurring of vision on the right side; whenever the patient looked at an object for more than a few seconds, the visual stimuli coming in from the patient's left field of vision tended to obliterate the visual stimuli which originated simultaneously from the right field. It was an example of the phenomenon of extinction. It seemed as if there was competition between the two sensory fields and resultant dominance by one of them. The theoretical implications of such "extinction" have been discussed in previous communications.^{4, 8}

CASE II.—A 42 year old man complained of "eye strain" of four weeks duration. Repeated perimetric examination failed to disclose a visual field defect. One day before admission to the Mount Sinai Hospital, there developed a mild disorder in language. Except for the slight dysphasia the neurological examination was negative. Routine perimetry again showed no defects, but with double simultaneous stimulation a defect was invariably found in the right half of the field of vision in each eye (right homonymous hemianopia). Subsequent clinical and laboratory studies strengthened the suspicion that a tumor was present in the left temporal lobe. A craniotomy performed three weeks later disclosed a large glioblastoma in the left temporoparietal lobes.

Comment.—In this case the method of double stimulation helped us to elicit signs of an additional defect. Although the speech symptoms

* This we call the "phenomenon of extinction."⁴ The phenomenon of extinction was described in the literature under different headings. Poppelreuter called it the phenomenon of "inattention,"⁵ Reider the suppression phenomenon⁶ and more recently Thiebaut and Guillaumat referred to it as "relative" or "pseudo" hemianopsia.⁷ In all these instances the method of double simultaneous stimulation was used.

had already suggested that a lesion might have been developing on the dominant side (left side in a right-handed individual) the demonstration of an hemiamblyopic defect on the right side made the localization of the lesion to the left side more certain.

The following case illustrates again how this manner of sensory testing might help us in localizing a cerebral lesion.

CASE III.—A 55 year old man was admitted to the Mount Sinai Hospital with severe headache and stiffness of the neck. These symptoms had suddenly appeared two days prior to admission. Routine neurological examination disclosed the classical signs of meningeal irritation, namely stiffness of the neck, positive Kernig and Brudzinski signs, photophobia and eyeball tenderness. There were also inconstantly positive Babinski sign and slight facial weakness on the left side. The standard visual field examination (with bedside perimetry) showed no impairment. When, however, objects were placed on both sides of the fixation point, he perceived the one on the right and not the one on the left. A left homonymous hemiamblyopia (extinction) was thus noted in each eye. The spinal fluid was uniformly bloody. The clinical diagnosis was spontaneous subarachnoid hemorrhage. Later in the course of the illness the field defect, originally present on bilateral stimulation, became apparent even with unilateral and single stimulations.

Comment.—The left facial weakness and Babinski sign pointed to involvement of the right motor region. In practice, however, such signs are not always of localizing value. The presence of a concomitant defect, especially a homonymous hemiamblyopia as demonstrated in this case by the method of double stimulation, certainly helped us to localize the lesion more accurately.

Double stimulation also aided us in our study of spinal cord cases.

CASE IV.—In a 59 year old woman with symptoms of a spinal cord tumor, the routine sensory status disclosed an ill-defined level in the midthoracic region. The sensory impairment in this case was slight and inconstant. When a single stimulus was applied in the lower thoracic or lumbosacral regions, she felt it as keenly as in the upper thoracic or face areas. On simultaneous application of one stimulus (pinprick) above and another below the sixth thoracic segment the patient reported that she perceived the upper but not the lower point of contact.

Comment.—This sort of suppression of a pain sensation ("extinction") below a given segmental level helped us to localize the lesion in the cord.

The method of simultaneous stimulation was also found useful in our studies of patients with profound mental disorders. In some patients with organic mental syndrome, single stimulations produced seemingly normal responses. Yet when the same patient was examined with the method of double simultaneous stimulation, a definite defect in sensation was found.

CASE V.—A 59 year old woman was studied at the Mount Sinai Hospital and later at Bellevue Psychiatric Hospital for a mental condition. The illness developed slowly over a period of three years. Except for mental changes characteristic of organic brain disease, the neuropsychiatric examination was negative. She was disoriented, confused and showed marked defects in memory. Despite this the patient was sufficiently cooperative to give reliable reports on sensory examinations.

The routine neurological status was essentially normal. There were no apparent alterations in any of the modalities. Nevertheless, with double simultaneous stimulation defects in cutaneous, visual and auditory perception were found and these defects were consistently present on the right side of her body.

Comment.—In examining a patient with an organic mental syndrome, it is sometimes difficult to evaluate the sensory status. In severe cases, the patient's responses to single stimulation tests may be varied or inconsistent. However, we found that in some of these patients with marked confusion, the method of double simultaneous stimulation showed alterations in sensory function which were consistent and localized to a given region of the body.

SUMMARY AND CONCLUSIONS

The method of double simultaneous stimulation may help us in demonstrating defects in function which routine sensory examinations may fail to bring out. The method has been most successful in patients with cerebral lesions and in some with lesions of the spinal cord. In patients with lesions of sensory roots or peripheral nerves, the method of double stimulation has no particular advantage, unless there was associated cerebral disease. However, we feel that it should be used in all cases since it is just as simple as single testing for the examiner, and actually less time consuming. For the patient, simultaneous stimulations are usually easier to compare with each other than several stimuli presented in succession. It is therefore concluded that the method of double (bilateral) simultaneous stimulation is useful and should be incorporated into the routine neurological examination.

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PSYCHOTIC SYMPTOMS MASKING THE ONSET IN CASES OF BRAIN TUMOR

SAMUEL BROCK, M.D.* AND BENJAMIN WIESEL, M.D.†

THE association of neurotic and/or psychotic symptoms with organic disease of the brain is common and generally recognized. At times the combination gives rise to clinical pictures of bewildering complexity, the more so since it is sometimes impossible to separate the basic organic components from the superimposed "functional."

One may encounter so-called "neurotic" symptoms, which on careful study prove to be due to organic disease of the brain. Thus, the neurasthenic symptoms of early general paresis and cerebral arteriosclerosis are well known. Then one encounters neurotic individuals who react with anxiety, fear, depression of mood, hypochondriasis and insomnia to the presence of organic disease whether of the brain or of other organs. There are still other instances in which mental symptoms of a psychotic nature occupy the foreground in the early months of illness, and lead to such diagnoses as "agitated depression," "schizophrenic behavior" and "involutional psychosis," until signs of organic brain disease "break through" and the important underlying organic disease becomes manifest. In the main, it would seem that these psychotic clinical pictures represent the reaction of an unstable personality to the presence of organic disease, although one cannot be sure that the organic cerebral process may not sometimes induce some of the symptoms by direct effect on "psychosomatic" mechanisms situated in the diencephalo-hypothalamic region.

We are not concerned at this time with mental symptoms directly due to organic disease of the frontal and/or temporal lobes, such as mental dullness, inattention, confusion, lack of insight, poor memory, undue jocosity, or visual, auditory or olfactory hallucinations, but rather with affective psychotic reactions usually occurring alone and unassociated with focal organic brain disease. When the clinician is faced with this type of case, his vigilance and skill are put to a severe test, but, as the following cases will demonstrate, he should be able to determine the presence of the focal brain disease sooner or later if he keeps a seeing eye and an open mind on the problem and properly uses and appraises laboratory data.

CASE I.—A wealthy industrialist about 62 years of age had built up a very successful business. He was a high-strung, neurotic, perfectionistic type of individual. In his past history one notes long-standing migrainous headaches and also attacks

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of vertigo of the Ménière type. For ten years or so he had been taking barbiturates in generous quantity to induce sleep. His family physician had observed him for many years and had treated him for many diverse neurotic complaints. There was a previous history of irregular periods of depression. Moreover, there was a curious domestic situation in that the patient paid a good deal of attention to a woman business associate, whose advice and companionship he sought over a considerable number of years. The patient seemed to have had little regard for his wife. She knew about "the other woman" but apparently offered no objection to the superior part which the latter played in her husband's life.

His present illness began in the summer of 1946. Certain near relatives, with whom he was associated in business, suggested that he leave more of the management of it to them so that he could rest more. He began to brood over this, believing that they wanted to supersede him; he became resentful and irritable. Depression of mood with brooding, anorexia, nausea, almost daily vomiting, sometimes induced by putting his fingers into his oropharynx, supervened. A well known gastroenterologist found no evidence of gastrointestinal disease by clinical or x-ray examination. His physician, an eminent internist, also failed to find any signs of visceral disease to account for the symptoms. Querulousness and dependence on various forms of medication developed. In December, 1946, he entered a hospital and was examined by an eminent neuropsychiatrist, who found no evidence of organic nervous disease. A diagnosis of agitated depression was made; four weekly electric shock treatments were administered and definite improvement set in; he slept and ate better and complained much less, but still was apathetic and disinterested. He would stay in bed and could only be encouraged to get up and make some steps with difficulty. At times he would collapse while on his feet, yet at other times he would walk about fairly well.

On January 9, 1947, he seemed stuporous; no focal signs could be demonstrated, but soon thereafter he ate better and vomited less often. He complained of heartburn. Two more electric shock treatments seemed to be of benefit. On January 24, 1947, an abnormal laboratory finding was noted; viz.: an increase in the spinal fluid protein to 102 mg. per 100 cc., together with "an increased pressure." On January 27, 1947, the spinal fluid was reexamined and the protein was still found to be elevated, i.e., 87 mg. per 100 cc. The pressure and other constituents were recorded as normal. Another laboratory finding was a sedimentation rate of 22 mm./hour on December 14, 1946, and 45 mm./hour on January 28, 1947.

On January 25, 1947, he was examined by a well known neurologist and an eminent internist. They concluded that his clinical picture was one of parkinsonism due to encephalitis; they found no evidence of metastatic cerebral neoplasm. When examined on January 29, 1947, by two other neurologists of long experience it was noted that his cooperation was poor, that he could not be engaged in conversation but understood and responded to single questions and commands. He was apathetic and depressed, complained of a pressure sensation in the back of the head, "heartburn and downheartedness." The mood change was reflected in his facial expression. There were no tremors, no spasticity, no reflex changes and no focal weakness. His left optic disk was normal, the right could not be seen because of a cataract; otherwise the cranial nerve innervations were normal. The visual fields could not be tested. He tended to lie curled up on one side. The weakness sometimes reported seemed of nonorganic nature, since at one time of day he would walk about freely and then a few hours later he would be unable to, or collapse if he tried. He stated that his vomiting was always self-induced in order to relieve either the heartburn or the nausea or the "pressure headache" in the "back of his head." He expressed no delusional or hallucinatory trends. At this time, the problem was quite puzzling; there seemed to be clinical features suggestive of an agitated depression; the diagnosis of parkinsonism on an acute encephalitic basis was not concurred in. The explanation of the increased sedimentation rate and the increased spinal fluid protein was not at hand.

The laboratory data were as follows

Röntgen study: Skull x rays Normal Lung x rays: January 10, 1947—congested left base, February 20, 1947—both bases congested, early broncho pneumonia There was no evidence of metastatic disease.

Electrocardiogram: Normal

Urine December 15 1946—Acetone two plus. Sugar appeared in the urine from time to time (due to intravenous glucose) December 21, 1946—Acetone two plus (appeared from time to time) Sugar three plus (due to intravenous feedings)

Blood Blood chemistry, December 16, 1946 urea nitrogen 18 mg., sugar 135 mg., calcium 9.9 mg., cholesterol 530 mg. Blood count normal range, hemoglobin 14 gm.; color index 1.0, white blood cells 13,500, normal distribution, further white counts showed about 13,000 cells. Blood Wassermann negative. Sedimentation Rate December 14, 1946 22, January 28, 1947, 45

Emesis, January 20, 1947 Brown liquid occult blood one plus, total acidity 56 free acidity 45

Spinal fluid January 24, 1947 Red blood cells 41, white blood cells 71, lymphocytes 70 per cent, chloride 712 mg., sugar 43 mg., protein 102 mg

January 27 1947 red cells 14, white cells 31, lymphocytes 100 per cent, chloride 749 mg., sugar 67 mg., protein 87 mg. Colloidal gold 0000000000, Pandy 1 plus February 21, 1947 No organisms found in spinal fluid.

In the next few days the headache and other symptoms continued On February 11, 1947, one of the neurologists attending the patient found flaccid weakness of the left extremities and possibly of the left lower face, forced grasping of the left hand, a questionable left Babinski toe sign and a left homonymous hemianopsia He lay in bed curled up on his left side His neck was not rigid There were no Magnus de Kleijn neck reflexes Both eyeballs were turned to the right (conjugate deviation) and could not be brought beyond the midline toward the left Despite very poor cooperation there was a suggestion of a sensory defect on the left half of the body In addition to the above mentioned symptoms, he complained of "smelling onions" A diagnosis of brain tumor, situated deep in the right temporofrontal region was made. On February 16, 1947, a neurosurgeon found him drowsy and biciping; his eyes were turned to the right and there was tenderness of the right temple and a change in the percussion note on the right side. A large diffuse glioblastoma was suspected

At operation the operator inserted a cannula deep in the right temporal lobe and at a depth of about 6.5 cm withdrew 15 cc. of a yellow fluid which clotted almost immediately The aspirated fluid contained shreds of tissue. An air injection was attempted, no air was found in the lateral ventricles The floor of the third ventricle appeared pushed down as if by a mass. On February 23, 1947, the neurosurgeon performed a right temporofrontal craniotomy The dura was tense Incision of the right temporal cortex disclosed an extensive deeply situated neoplasm, firm in some, cystic in other parts. Some thrombosed vessels were found. The neoplasm extended to and probably beyond the midline. A large part was removed The cellular structure suggested that the tumor may have arisen from the pineal gland

Comment.—This case is an unusual example of organic brain disease which was masked for a time by mental symptoms which seemed to be part of a nonorganic psychotic mental state. It is difficult to separate the early symptoms due to the abnormal personality reaction from those due to the presence of the brain tumor. The temporary improvement under electric shock therapy, the high spinal fluid protein and the increased sedimentation rates are noteworthy. The omission of electroencephalographic studies was due partly to the fact that by the time the patient's signs pointed to focal cerebral disease, the diagnosis

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was obvious. Electroencephalography might have put us on the track earlier if it had been used.

CASE II.—A man of 38 years, a doorman in a night club, was first seen on October 20, 1944. He had been married for four years. He had always been a rather withdrawn, timid person, given to fainting easily. Early in August 1944, he began to experience dizziness, nausea and the vomiting "of bile" on arising. A gastrointestinal x-ray series was negative. A nose and throat specialist treated him for "adenoidal abscesses" without benefit. In the first three weeks of October 1944, the dizziness had increased and he felt as if he might fall from time to time; a feeling of general weakness was also complained of. At times his head felt "like a piece of ice" and a weight "behind my neck passed to my chest and I could not breathe easily." Then he would experience dizziness. Sometimes a sensation of "needles in all the fingers" was felt. Sleep was broken at times by coughing and expectoration. He could not shave himself because he could not throw his head back and he suffered a peculiar reaction when light struck his eyes. Of late sexual desire had disappeared. His wife remarked on the fact that "now he sits home and stares out of the window and doesn't talk much; he seems changed."

The family history was of importance. One sister has been in a mental hospital for ten years because of dementia praecox. There are five brothers; one who accompanied the patient was a very neurotic individual who interfered with the examination of his brother on numerous occasions to describe his own neurotic symptoms. Two unobserved brothers were said to be "queer" fellows.

At the time of the initial examination the neural status was entirely normal except that the patient kept his neck somewhat flexed. He was able to correct this posture without difficulty. He seemed somewhat depressed and irritable; there were no abnormal mental trends or defects.

He was admitted to a hospital on October 23, 1944, and was observed on several occasions during the next ten days. He ate well, offered little in the way of spontaneous complaints, seemed depressed, moody, quiet and rather seclusive; there seemed to be some poverty of affect but this varied from time to time. There were no abnormal trends or evidences of gross mental defect. A number of neurological examinations were negative, though the flexed posture of the cervical spine was noted several times. He complained of dizziness on change of head position, such as on arising, but on several occasions he was asked to arise quickly and no dizziness was complained of. On two occasions, the examiner rotated the patient's head rapidly from side to side several times without eliciting dizziness. Neither neck ache nor headache were complained of. On the day before discharge he had taken a long three hour walk with his brother and when he returned he said he felt "fine." Because of his family history and his personality reaction the situation was regarded as probably an early mental disorder (schizophrenia?) but no conclusion had been reached.

During the patient's hospital stay, the blood Kline was negative; so was the urine examination except for an occasional granular cast. The blood sugar was 89 mg. per 100 cc. Previous blood counts were normal. X-rays of the cervical spine were negative except for some lipping of the anterior margins of the 6th and 7th cervical vertebrae.

Because he did not improve on a variety of medications and seemed to grow more shut-in and irritable, the patient entered a well known mental hospital in November 1944. The chief complaints then were "nervousness, irritability, mild depression, weakness of will, dizziness, headaches, insomnia, and physical complaints centered around the stomach."

On November 25, 1944, the patient's mental status was as follows: He seemed irresolute, indecisive, and showed a markedly depressed emotional tendency. He was found to be a healthy appearing person, but complained of various painful sensations, as a sense of pressure in the head and neck, gastrointestinal discomfort,

cardiac palpitation, breathlessness, flatulence, fullness in the stomach, constipation, twitching of muscles weakness of limbs general fatigability, inability to sleep, lack of concentration, inability to do things depression irritability and dizziness He stated that every morning when he awoke he had the feeling of dizziness When he stood he felt dizzy and had a peculiar sensation in his limbs He suffered from a feeling of anxiety and thought that he was about to lose consciousness He was very interested in the mental examination The stream of mental activity was relevant, coherent and the psychomotor activity was normal He talked a good deal about his physical condition He seemed to be in a state of tension and emotional instability and appeared somewhat fearful and apprehensive There was no dissociation of affect and thought, no inadequate or incoordinate affect, perplexity or apathy yet he had lost interest in certain of his former activities, viz. movies, games and social gatherings He showed a continuous preoccupation with his state of health and of various organs He was convinced that his gallbladder was seriously impaired No delusions or hallucinations could be elicited His sensorium was clear He was oriented as to time, place and person He had a fair knowledge of current events and his memory for remote or recent events was not impaired His grasp of general information was fair and retention was good The seven minus one hundred test was well performed Speech and writing were normal Judgment might have been somewhat defective; he was incapable of making plans for the future He felt that he was only physically sick and that there was nothing wrong with him mentally

Later the patient complained of severe pains in the back of his neck, cardiac palpitation gastric disturbances nervousness, insomnia and dizziness Neurological examination disclosed no new findings The heart was normal The blood pressure was 140/65 X-rays of the chest entire spine and skull were normal, so were blood counts, blood chemistry and urine examinations Early in December 1944, he was given one electric shock treatment. However, in contrast to the experience in Case 1, this form of treatment caused a speedy and remarkable increase of organic signs Directly following the one and only electric shock treatment he complained of severe occipital headache, and he vomited several times Horizontal nystagmus to the left bilateral papilledema (4 diopters) with retinal hemorrhages in both eyes, right more than left, posturing of the head with chin tilted to the left, a broadened ataxic gait a positive Romberg sign bilateral limb ataxia hypotonic deep reflexes and bilateral Babinski toe signs pointed to a midline posterior fossa tumor

An electroencephalographic study made on November 27, 1944, showed "Resting (Fasting) Occipital Region Well defined alpha activity Frequency 9 per sec. Continuity about 50 per cent Regularity Mixed with episodes of slow activity (4 per sec. max amp 40 micro volts) This slow activity predominates occasionally in the left occipital region Max Amplitude 40 micro volts Anterior Leads Same type of alpha activity with some slow waves in the monopolar leads, but less than in the occipital region Hyperventilation Increase of slow activity in the occipital region Impression This is an abnormal record showing bursts of moderately slow waves in both occipital regions" Repeated examinations are suggested in order to localize the abnormality better

The patient was seen again on December 10, 1944, when the following note was made "The patient's unsteadiness of gait has increased, the pressure and ache of the back of the neck is more pronounced There is difficulty in swallowing and nausea and salivation In the past two weeks there had been occasional attacks of diplopia Physical signs show instability in gait with positive Romberg The patient postures his head somewhat forward now There is slight left finger to nose ataxia but no adiadochokinesis There is some left past pointing In the cranial nerve sphere, there is marked papilledema and horizontal nystagmus There is some increase of the deep reflexes in the left upper limb but there is also increase of the right biceps jerk The abdominal reflexes are diminished on the left There is

no Babinski toe sign and there are no sensory changes. There are no new mental signs, no aphasia, no apraxia."

New laboratory studies revealed normal blood counts and urine. Ventricular fluid (December 12, 1944) showed 4 white blood cells, protein 16 mg. per 100 cc. and normal colloidal gold curve and a negative Wassermann in various dilutions. X-rays of the skull (December 11, 1944) revealed slight atrophy of the sella turcica suggestive of an expanding intracranial lesion.

A ventriculogram (December 12, 1944) revealed marked dilatation of the lateral and third ventricles; the aqueduct and fourth ventricle were also dilated; there was a soft tissue mass projecting into the fourth ventricle from its floor obliterating the lower half. The basal cisterns were not filled. Impression: Fourth ventricle tumor, probably ependymoma.

On December 11, 1944, an electroencephalogram was essentially normal.

On December 12, 1944, the neurosurgeon encountered "a large firm tumor occupying the fourth ventricle extending up to the aqueduct; the upper four-fifths was removed without difficulty, the lowest part embraced the posterior inferior cerebellar artery of each side." The growth proved to be an ependymoma. Following the operative removal of the growth, x-ray treatment was begun. A slow convalescence set in. He was able to resume work in September 1945. By March 1946, he was found to be fairly well and working as usual. In July 1946, the only symptom was occasional dizziness on quick movement.

In July 1947, the patient's family physician reported that he still acts a bit peculiarly; i.e., he is quiet, seems to have some poverty of affect and is impotent. He finally died from the effects of the brain tumor in November, 1947.

Comment.—In this case we were dealing with a man possessing an abnormal personality (schizoid type). His family history suggested poor germ plasm from the standpoint of mental disease. The developing clinical picture of a brain tumor was obscured by his personality reactions. In retrospect, the flexed posture of his neck, the increasing neckache and dizziness should have pointed the way to an earlier diagnosis but these symptoms were lost in the welter of other less important symptoms. One electric shock treatment brought to the fore very definite organic signs of brain disease.

CASE III.*—A 38 year old white married woman, the mother of three children, had always been a nervous and rather hypochondriacal type. In the previous history, there were three operations for pilonidal cyst and a gallbladder operation. Her menstruation had become irregular in the spring of 1947, and in September 1947 she became irritable, depressed and more hypochondriacal. She complained of headaches and limb pains. Treated with estrogenic hormones, her menses returned. In early June 1947 her menses were again irregular and she became listless, apathetic, very careless and complained of extreme fatigue. She wanted to stay in bed most of the time and on two or three occasions urinated in bed. Pains and aches in the shoulders, back and abdomen were mentioned; she worried about herself and expressed the fear that she was going insane; she thought that people stared at her for unknown reasons; she couldn't sleep and "things were not worth while." She slowed down greatly and had to be forced to eat. At times she wept, at other times there was inappropriate laughter.

Neurological examination in June 1947, disclosed no abnormalities. The patient's behavior was as above-described; she was psychically retarded, would stare ahead for long periods, seemed emotionally inadequate, apathetic, uncooperative, and

* We are indebted to Dr. Foster Kennedy for permission to report this case.

now and again would laugh for no obvious reasons. In the first week of July 1917 she received three electric shock treatments. After the first two treatments she was considerably more alert. After the third (July 6) she appeared somewhat wobbly and vomited. On July 7 she became unresponsive, mumbled and slowly lapsed into coma. She was taken to Bellevue Hospital, New York City. Examination disclosed the following: She was in coma but answered to her name and could be aroused by supraorbital pressure. Her temperature was 100.2° F., pulse rate 60, respirations 20, with occasional changes in depth suggestive of Cheyne-Stokes respiration. Her blood pressure was 110/70. She would yawn, pick her nose with her left hand, and would rub and pick the bedclothes. Her right pupil was irregular and of greater size than the left. They reacted to light. There was bilateral papilledema (2 to 3 diopters), a divergent strabismus, an impairment of left lateral gaze and equally active corneal reflexes. The neck may have been slightly rigid. There was no paralysis, but muscle tone was increased in the lower limbs, more so on the left, and the deep tendon reflexes were more active on the left. The abdominal reflexes were absent. The Babinski toe sign was present on both sides. The patient was incontinent of urine. A right frontal neoplasm was suspected.

On July 7, 1917, the neurosurgeon found a drowsy woman barely able to talk. There was slight widening of the right palpebral fissure and the right pupil was slightly larger than the left. There was bilateral papilledema, bilateral forced grasping on tactile stimulation of the palms, bilateral Babinski toe sign, left more than right, and deep reflexes more active on the left. The percussion note was flatter on the right half of the skull. Skull films showed no evidence of meningioma or aneurysm. The dorsum sellae looked atrophic as if from chronic increased intracranial pressure. It was believed that the patient was suffering from a deep right frontal glioblastoma multiforme with probably bifrontal extension.

Under local anesthesia a right frontal trephination was made. The brain was under extreme pressure and bulged forcibly through the dural opening. A brain needle met rubbery resistance at a depth of 3 cm. and aspiration biopsy from a depth of 4.5 cm. yielded purplish gelatinous tissue typical of a glial tumor. The trephine opening was enlarged so that a small transcortical incision could be made to inspect the tumor. The nature of the tumor was verified and some of it removed for biopsy and decompression. The dura was not closed as brain tissue herniated through the dural opening. The condition of the patient was improved after the operation. She spoke rationally, was not drowsy and could move all four extremities and showed no facial weakness. The forced grasping was no longer pronounced. There was bilateral Babinski toe sign.

Comment.—As in the two previous cases, a group of mental symptoms obscured the onset of what proved to be a brain tumor. Electric shock treatment, as in Case II, brought the organic signs to the fore.

CASE IV.—This concerns an attorney of 31 years of age, who was seen in February 1916. One year prior he had married a "superior woman" of wealth. His parents had been poor. Late in 1915 he began to develop self-accusatory trends; he thought he was not good enough for his wife and he blamed himself for marrying her. Though he was obsessed with his feeling of inferiority, he became very critical of his wife. His mood was one of depression and he felt "inadequate and emotionally weak." Though he continued his court work he could not concentrate and he felt that he "might be going insane." He felt "trapped," doubted the "value" of having married, slept poorly, thought only of himself, and even contemplated suicide but was "afraid to hurt" his pregnant wife. At times he was agitated.

The neurological examination was normal, including examination of the fundi. Eight electric shock treatments brought about a very great improvement by the middle of March 1916. The patient was a little confused and no longer agitated, though given to ruminating and thinking of suicide. Later in March the tendency

to rumination continued and some degree of agitation reappeared. Two more electric shock treatments brought about better sleep and appetite, and though less depressed he seemed somewhat hypomanic and silly. Four more electric shock treatments were given and he felt much better. The depression had lifted; he played cards, wanted to resume work and was no longer afraid to meet people. The improvement was short-lived, for in two more weeks he felt he was going insane, his tendency to rumination returned and he discussed the "legal concepts of sanity, insanity and the semantics of sanity." He felt he enjoyed his illness and was hypochondriacal.

Six more electric shock treatments were given and by the end of April 1946, he felt his illness was at an end; the ruminations had ceased and the depression had disappeared, but he was markedly confused. Two more electric treatments were given and "I feel fine"; he was garrulous and expansive. His baby was born about May 10 and he began to worry again but none the less he "felt fine." He seemed to lack judgment: his reactions seemed superficial. For about six weeks there was no change but by mid-June, 1946, a relapse set in: he became obsessive and preoccupied again; he was overtalkative, circumstantial, rambling and introspective. There was some mental retardation. A shallowness of affect was noted. Now neurological examination revealed papilledema, an inequality of pupillary reaction, the right being greater; the left abdominal reflexes were diminished, the left plantar response equivocal and there was a slight left facial weakness. A right-sided brain tumor was suspected.

The electroencephalogram was considered to be within normal limits.

A ventriculogram was done which showed a shift of the ventricles to the left with compression of the anterior portion of the temporal horn on the right. A right frontolateral craniotomy was carried out and a large infiltrating tumor was encountered in the right temporal lobe. It was grayish-pink in color, infiltrated the brain tissue and was judged to be the size of a plum. The pathological report described a transitional glioblastoma.

Comment.—In this patient, an agitated depression (with other manic-depressive features) constituted the initial illness. It responded favorably to electric shock treatment. Yet about eight months after onset, signs of a brain tumor appeared and operation confirmed its presence.

SUMMARY AND CONCLUSIONS

Experienced psychiatrists, especially those in up-to-date mental hospitals, are well aware of the incidence of brain tumor in the insane (McIntyre and McIntyre,¹ Larson,² Hoffman³ and Jameison and Henry).⁴ As our cases illustrate, there is no psychiatric syndrome characteristic of brain tumor—a point made by Jameison and Henry. Neurologists have also laid stress on the coincidence of psychotic symptoms and tumor of the brain (Schuster⁵ and Baruk⁶). By repeated neurological examinations and the use of electro- and pneumo-encephalography one should be able to recognize these cases early and save oneself the embarrassment of faulty diagnosis, inappropriate treatment and incorrect prognosis. In this small series of four cases, the temporal and/or frontal regions seemed to be the usual site of the tumor; however, the midline posterior fossa ependymoma (Case II) shows that other parts may be affected.

The matter of electric shock treatment is of interest in three of the cases. It seemed to be of temporary benefit in Cases I and IV, whereas in Cases II and III it caused an immediate increase of symptoms and signs and the diagnosis became apparent.

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TULAREMIC MENINGISM AND SEROUS MENINGITIS

A Report of Twenty-Eight Cases Observed During an Epidemic of Tularemia Transmitted by Insects in Settlements of Deportation Asino and Jaja, Siberia, USSR.

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IN a previous paper¹ a report was made of 121 cases of tularemia observed in settlements of deportation Asino and Jaja, Novosibirsk Province, Siberia, Soviet Russia, in the Summer of 1941. One hundred fifteen cases out of the total 121 belonged to the ulceroglandular form and 114 cases were confirmed by serologic and/or an allergic test for tularemia. No direct contact of the infected persons with the rodents could be traced in any of these cases. The source of infection was the water rats (*Arvicola amphibius*) populating the shores of the adjacent river, Czulym, shortly after the late spring floods. The infection was transmitted to man by small insects, probably by mosquitoes. The epidemic was rather benign in nature and the mortality rate was only 1.7 per cent (two deaths out of 121 cases). A striking clinical feature of this epidemic was the uncommon location of the primary tularemic focus. In eighty-one, or in two-thirds of the cases, this was on the leg or the foot and was associated with a regional inguinal or femoral lymphadenopathy.

Another outstanding feature of this epidemic was the initial and transient involvement of the meninges in a form of meningism or serous meningitis. These observations warrant publication since no similar information could be found in the world literature.

MENINGEAL INVOLVEMENT IN TULAREMIA

Tularemic meningitis is a very rare complication of tularemia. Only six fatal cases of purulent tularemic meningitis are recorded in the American literature.^{2, 3, 4, 5, 6, 7} In all of these cases purulent meningitis developed between the fourth and the tenth day of the disease and caused death within three to five days. Pullen and Stuart,⁸ however, observed a 10 year old boy with purulent tularemic meningitis who recovered. The rarity of these cases is striking, if one compares them

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with the total number of tularemic cases reported in the United States (more than 10,000).

Severe headache and vomiting are classical initial symptoms of tularemia. However, no stress was ever laid, even in the most important classical descriptions of this disease, on the relation of these signs to meningeal irritation. Francis⁹ in his study of tularemia states: "Severe meningeal involvement was indicated in cases manifesting delirium and stupor." Pullen and Stuart⁸ have noted "several instances of stupor, meningism and other signs of possible central nervous system involvement" among their 225 cases of tularemia. Finally Kavanaugh¹⁰ noted delirium in six cases among his 123 cases of tularemia and acute mania in one.

There is no record of an epidemic of tularemia with the high incidence of signs of meningeal involvement as the one reported here. The cases of tularemic meningeal involvement, which were observed during the epidemic in Asino and Jaja, differ in many respects from the reported cases of tularemic meningitis:

Meningeal involvement manifested itself most frequently by signs of meningism and at times by signs of serous meningitis or meningo-encephalitis. Purulent meningitis was not found in any of our cases. Meningeal involvement was an initial sign of acute tularemic invasion, and not a terminal complication. The involvement was usually transient and regressed during the course of the disease. It was very common in patients observed during the epidemic of tularemia in Asino and Jaja. It was found in almost one-half of these cases (in twenty-eight of a total of fifty-eight hospitalized cases).

The meningeal involvement was intimately associated with the other essential signs of ulceroglandular form of tularemia. Frequently the diagnosis was possible on the basis of the *characteristic tetrad of signs* shown by the early hospitalized patients:

1. Signs of meningism, i.e., stiff neck, positive Kernig and Brudzinski signs, headache and vomiting.
2. High fever.
3. Painful regional lymphadenitis usually located in the inguinal or femoral area.
4. The primary focus consisting of a characteristic papule or pustule surrounded by a small area of infiltration and usually located on the lower leg or on the foot.

The diagnosis later was usually confirmed by spinal tap, agglutination reaction, allergic intradermal test (see Table 3). The course of the disease was benign, for recovery occurred in all but one case.

Among these patients were sixteen children below 16 years of age, seven young adults (between 16 and 20 years) and five adults between the ages of 30 and 40 years. The female sex predominated: twenty-two female and six male patients were observed. Meningeal involvement

TABLE 1

CLINICAL SIGNS OF MENINGEAL INVOLVEMENT IN TWENTY-EIGHT CASES OF TULAREMIA

Case No.	Sex	Age	Site of Primary Focus	Duration of Illness Prior to Admission in Days	Signs of Involvement of Central Nervous System					Remarks
					Duration of CNS Symptoms in Days	Stiff Neck	Kernig Sign	Brudzinski Sign	Stupor (+) or Coma (++)	
1	F.	19	Leg	2	2	++	++	+	++	{ Delirium Hallucinations
2	F.	10	"	2	3	++	++	+	++	
3	F.	23	"	7	3	++	++	++	+	
4	F.	14	"	2	6	++	++	++	++	{ Delirium Hallucinations Hyperkinesia Hypocinesia Post. Babinski
5	M.	9	"	2	3	++	++	++	++	
6	M.	13	"	1	3	++	++	++	++	
7	F.	32	"	4	1	++	++	++	++	{ Delirium Hypocinesia Post. Babinski
8	F.	16	"	1	2	++	++	++	++	
9	F.	16	"	1	9	++	++	++	++	
10	F.	16	"	7	4	++	++	++	+	{ Transient aphasia Convulsions Delirium
11	F.	32	"	3	2	++	++	++	++	
12	F.	17	"	3	8	++	++	++	++	
13	F.	40	"	4	4	++	++	++	++	{ Delirium Delirium (See Report)
14	F.	10	"	2	5	++	++	++	++	
15	F.	24	"	3	1	++	++	++	++	
16	F.	20	"	3	2	++	++	++	++	{ Delirium Hyperkinesia Positive Ba- binaki
17	M.	13	"	2	1	++	++	++	++	
18	F.	16	"	3	2	++	++	++	++	
19	F.	39	"	3	5	++	++	++	++	{ Delirium Hyperkinesia Positive Ba- binaki
20	F.	7	"	3	5	++	++	++	++	
21	F.	13	"	3	5	++	++	++	++	
22	M.	7	"	3	5	++	++	++	++	{ Delirium Hyperkinesia Positive Ba- binaki
23	M.	14	"	3	5	++	++	++	++	
24	M.	21	"	3	5	++	++	++	++	
25	F.	16	Foot	2	1	++	++	++	++	{ Delirium Hyperkinesia Positive Ba- binaki
26	F.	14	"	2	3	++	++	++	++	
27	F.	22	"	2	5	++	++	++	++	
28	F.	22	Forearm	5	2	++	++	++	++	

was found only in patients with ulceroglandular form of tularemia. Twenty-seven out of twenty-eight meningeal cases of tularemia (i.e., 96 per cent of cases) had the primary focus of tularemic invasion on the leg or on the foot.

The twenty-eight cases of tularemia with meningeal involvement are listed in Tables 1, 2 and 3.

The symptoms of *meningism* found in cases of tularemia with central nervous system involvement were: stiff neck, positive Kernig sign, positive Brudzinski sign, stupor or coma lasting several days, severe headache preceding and following the period of stupor and coma, increased pressure of the cerebrospinal fluid, improvement of all symptoms after spinal tap, hypersensitivity of the skin, and bradycardia. The last two signs were inconstant.

Spinal tap was done in twenty-four of the twenty-eight patients showing signs of meningeal involvement (Table 2). In the remaining four cases the symptoms of meningism were too slight to offer an indication for lumbar puncture.

The pressure of the cerebrospinal fluid upon spinal tap was increased in all cases, so that the fluid spurted out in form of an arc (+++), straight stream (++) or by fast drops (+). Owing to the lack of equipment in the Siberian settlement hospital, manometric readings of the cerebrospinal pressure could not be recorded. In most cases 30 to 40 cc. of fluid had to be removed before the pressure returned to normal. In severe cases it was necessary to remove 60 to 70 cc. of fluid before the flow by drops started.

The cerebrospinal fluid was transparent, clear and watery in all the cases. The protein content was below 60 mg. per 100 cc. in two-thirds of the cases and up to 100 to 160 mg. per 100 cc. in the remaining one-third of the cases. The Nonne-Appelt reaction was usually negative. It was slightly positive in a few cases showing increased amount of protein in cerebrospinal fluid. The cell count was normal in most of the cases. The chlorides, glucose and colloidal gold curve, as well as the culture of the cerebrospinal fluid and animal inoculations were not done because of insufficient facilities.

The meningism usually appeared during the early days of the disease. All of these patients were brought to the hospital with obvious meningism. The duration of the meningeal symptoms observed in the hospital was one to three days in sixteen patients, four to six days in eight patients, and seven to nine days in four patients, with an average of four days. Only eleven patients were brought to the hospital within the first forty-eight hours of the disease and seventeen were hospitalised between the third and seventh day of the illness. Since most cases just mentioned showed a history of meningism for one or two days prior to the patient's admission, the real duration of tularemic meningism was longer, with an average of about five or six days at least.

TABLE 2

SPINAL TAP IN CASES OF TULAREMIA WITH MENINGEAL INVOLVEMENT

Case No	Spinal Tap				
	Pressure *	Cc Removed	Transparence	Proteins gm per 100 cc	Cells per cu mm
1	++	40	Clear	0.02	4
2	+	20	"	0.16	29
3	+	30	"	0.02	3
4	+	25	"	0.10	5
5	++	30	"	0.13	5
6	++	35	"	0.06	6
7	+	20	"	0.04	2
8	+++	60	"	0.10	3
9	+	17	"	0.10	5
10	++	25	"	0.06	6
11	—	—	—	—	—
12	++	10	"	0.04	0
13	++	20	"	0.06	3
14	++	26	"	0.07	5
15	++	20	"	0.10	7
16	+++	25	"	0.06	2
17	—	—	—	—	—
18	++	20	"	0.06	3
19	—	—	—	—	—
20	—	—	—	—	—
21	+++	60	"	0.03	?
22	++	40	"	0.10	?
23	++	70	"	0.12	10
24	+++	70	"	0.06	13
25	++	20	"	0.02	0
26	++	15	"	0.06	1
27	++	60	"	0.06	?
28	++	30	"	0.013	3

* The pressure is indicated in the following way

(++++)—the cerebrospinal fluid spurted out in form of an arc,

(++) —the cerebrospinal fluid spurted out in form of a straight stream,

(+) —the cerebrospinal fluid ran by fast drops

The following case report exemplifies the usual course of these cases

A boy of 15 years of age (Case No. 6) became ill on August 6, 1911, with signs of severe headache, vomiting, chills, prostration, fever up to 101° F., and pain in the right inguinal area. On the next day he was brought to the hospital in a comatose condition with stiff neck, positive Kernig and Brudzinski signs, temperature 101° F., pulse 90 and enlarged, firm, not adherent inguinal lymph glands on the right side. A typical papulopustule was present on the lower part of the right leg surrounded by a small red area of inflammation. The lungs and heart were normal on percussion and auscultation. The spleen was not enlarged and the liver was palpable for one fingerbreadth below the costal margin.

Spinal tap on August 7, 1941, revealed a cerebrospinal fluid flowing under increased pressure in the form of an arch. Thirty-five cc. of a clear and transparent fluid were drawn; it contained 60 mg. of protein per 100 cc. and 5 cells per 1 cu. mm. The Nonne-Appelt reaction was negative.

The white blood cell count was 6000 per cu. mm. with neutrophils 58 per cent (immature cells 14 per cent), lymphocytes 37 per cent, monocytes 5 per cent.

The intradermal allergic test, which was negative on the sixth day, became positive on the twentieth day of the disease. The agglutination reaction for tularemia was strongly positive in the titer of 1:400 on the twentieth day of the disease.

The general condition of the patient improved slightly after the spinal tap, which restored consciousness. On August 8th, 1941, marked hypersensitivity of the skin was noted. The patient was restless and had hallucinations. The stiff neck, Kernig and Brudzinski signs persisted three days longer. The fever persisted for ten days, varying between 102 and 104° F.; this was followed by a low grade elevation of temperature for an added two weeks. The primary focus was visible until August 17, 1941, at which time it became dry. The lymph glands were very painful at the beginning but gradually became painless, hard and decreased in size at the end of the third week. After six weeks they became normal.

The patient was discharged from the hospital in a good general condition on September 17, 1941.

Because of absence of inflammatory signs in the cerebrospinal fluid most cases of tularemia with meningeal involvement should be classified as tularemic meningism. However, some of our cases showed meningeal symptoms of a longer duration (eight to ten days), an increase of the protein content and slight increase in cells in the cerebrospinal fluid. These cases should be classified as *tularemic serous meningitis* rather than meningism (cases 2, 4, 5, 8, 9, 15, 22 and 23).

Four of the cases showed distinct signs of cerebral lesion in addition to meningeal involvement, so that *meningo-encephalitis* was suspected in these cases. The existence of this type of lesion in tularemia was proved by Hartman.¹¹ In addition to the usual meningeal signs and symptoms these four cases (Nos. 10, 16, 24 and 28) showed all or some of the following signs: hallucinations, restlessness or somnolence, hyperkinesia, excitation of the motor area of the cerebral cortex in the form of transient convulsions of short duration, positive, usually unilateral, and transient Babinski, incontinence of urine and feces, hippus of pupils, or wide pupils very slowly reacting to light. Three of these patients recovered, and one (No. 24) died.

The report of this *fatal case of tularemic meningo-encephalitis* follows:

A boy of 14 years of age (Case No. 24) became ill on August 28, 1941 with severe headache, pains in the left inguinal area, chills, and high fever. On the next day he lost consciousness according to the report of his father. In spite of this turn in his condition he was kept at home for three more days and was brought to the hospital only on the fifth day of the illness, i.e., on September 2, 1941.

On admission he was in coma and showed a stiff neck and strongly positive Kernig and Brudzinski signs. The temperature was 104° F. and the pulse was 86. Near the left malleolus a typical primary tularemic focus in form of a papulopustule was present. It was surrounded by a zone of infiltration. In the left inguinal area enlarged and hard lymph glands were present. The heart, lungs and abdomen

were normal on physical examination. The leukocytes numbered 4000 per cu. mm. The differential count was: neutrophils 73 per cent (immature cells 24 per cent), lymphocytes 23 per cent, monocytes 4 per cent.

The spinal tap revealed the cerebrospinal fluid flowing under a high pressure in form of an arch. Forty cc of a clear and watery fluid were removed. The protein content was 60 mg per 100 cc., the Nonne-Appelt reaction was negative and 4 cells were found per cu.mm.

The treatment was symptomatic, since serum was not available and streptomycin was unknown at this time.

On September 3, 1941 the patient was semiconscious, restless and had hallucinations. The temperature remained 104° F., the pulse rate was 88. The skin was hypersensitive, the neck was stiff, and Kernig and Brudzinski signs were positive. The left inguinal lymph glands were very painful.

On September 4, 1941 the patient was still semiconscious and hallucinated. He showed hyperkinesia and developed incontinence of urine and feces. The signs of meningism persisted. The temperature was 103° F., and the pulse 90. The infiltration around the primary focus increased. At the site of the pustule a small ulceration was formed. The intradermal reaction with tularin was strongly positive. The treatment consisted of small doses of barbiturates and hypertonic dextrose solutions injected intravenously.

A second spinal tap showed increased pressure of the cerebrospinal fluid which spurted in a form of an arch. Sixty cc. of transparent and watery fluid were removed when the cerebrospinal fluid started to run in fast drops. The protein content of the cerebrospinal fluid was 60 mg per 100 cc., the Nonne Appelt reaction was negative, and 6 cells per cu.mm. were found.

On September 5, 1941, the patient developed hippus of the pupils, hyperkinesia, jerky tendinous reflexes and lost consciousness. The meningeal symptoms slightly decreased, but incontinence of urine and feces persisted. The temperature was 103° F. and the pulse 100.

On September 6, 1941, the patient was in deep coma. The pupils were large showing intermittent hippus. Pallor of the face and cold sweat of the extremities were present. The patient was hyperkinetic in spite of the coma. Involuntary movements of hands of a grasping character (carphology) was noted. The stiffness of the neck was less pronounced and Kernig and Brudzinski signs were less marked. The temperature was 101° F., the pulse was 120 and threadlike. The left inguinal lymph glands were very large and firm.

In the evening of the same day agony started and by next morning (September 7, 1941) the patient died. Autopsy consent was not granted.

COMMENT

The involvement of the central nervous system in tularemia seems to be due to a certain neurotropic affinity of the *Bacillus tularensis*. If the affinity is small and the degree of the invasion of the central nervous system is slight, the involvement of the central nervous system manifests itself in severe headache, vomiting and disturbance of consciousness. These constant initial signs of acute tularemia indicate slight meningeal irritation. If the intensity of attack in regard to the central nervous system is greater, meningism or serous meningitis with its usual signs and symptoms results. If the attack is more severe meningo-encephalitis may result. Finally in the most profound form, when the affinity of *Bacillus tularensis* for the nervous system and the virulence of the strain are still greater, fatal purulent meningitis may develop,

TABLE 3

AGGLUTINATION TEST, ALLERGIC INTRADERMAL TEST, WHITE BLOOD COUNT AND SEDIMENTATION RATE IN CASES OF TULAREMIA WITH MENINGEAL INVOLVEMENT

Case No.	Agglutination Test in Serum for Tularemia		Allergic Intradermal Test for Tularemia		White Blood Cell Count					Sedimentation Rate		
	Day of Illness	Result and Titer	Day of Illness	Result	Day of Illness	Number per cu. mm.	Neutr. %	Lymph. %	Monoc. %	Eosin. %	Day of Illness	Mm. in 1 hour
1	15	+(1:200)	12	+	5	4400	52	40	8	0	5	18
2	22	+(1:400)			15	28000	?	?	?	?	24	8
3	14	—	15	+	13	3800	69	26	2	3	12	17
4	20	+(1:400)	7	+	5	6400	57	35	8	0	14	7
5	20	+(1:400)	6, 20	+	5	5600	68	20	12	0	12	52
6	20	+(1:400)	20	+	4	6000	58	37	5	0	6	22
7	15	+(1:200)	6	—	5	3800	75	18	7	0	14	51
8	16	+(1:400)			5	6200	76	12	12	0	2	15
9	17	+(1:400)			3	3200	72	22	6	0	8	21
10	12	+(1:100)			10	3200	63	31	6	0	10	31
11	9	—	10	+	6	3600	64	28	8	0	3	19
12	14	+(1:400)	4	+	8	10200	51	43	6	0	9	18
13	16	+(1:200)			7	6200	56	34	10	0	7	28
14	15	+(1:200)			7	6200	57	31	11	1	7	28
15	15	+(1:100)			11	11400	38	57	5	0	11	22
16	15	+(1:100)	9	—	3	6800	61	35	4	0	3	32
17	17	+(1:400)	13	+	6	8200	72	18	8	0	5	16
18	13	+(1:100)	5	+	4	8300	75	20	5	0	28	15
19	14	+(1:200)	6	+	6	8300	77	18	3	2	6	19
20	19	+(1:400)	3	+	3	3800	86	12	2	0	3	14
21	14	+(1:800)	8	+	4	7400	66	27	7	0	5	26
22	14	+(1:50)	14	+	17	11700	50	46	4	0		
23	13	+(1:100)										
24	24		8	+	7	4000	73	23	4	0		
25	15	+(1:50)	6	+	5	7000	65	23	12	0	13	9
26	19	+(1:400)	16	+	4	4800	65	29	6	0	7	24
27	16	+(1:800)	11	+	6	12000	66	26	8	0	6	32
28	20	+(1:200)				6000	66	28	6	0		

It is not clear upon what factors invasion of the central nervous system by the *Bacillus tularensis* depends. The virulence of the strain and its various organotropic affinities seem to have some importance. Apparently the sex and the age also predispose to more or less severe involvement of the central nervous system. Children accounted for one-half of the cases of tularemic meningism; this is analogous to the frequency of meningism in children in other infectious diseases (pneumonia, etc.). Also females seem to be susceptible to the involvement of the central nervous system in tularemia. Meningism occurred four times as often among females as among males in this epidemic; the general ratio between females and males was 2:1.

SUMMARY

A short epidemic outbreak of tularemia consisting of 121 cases was observed in settlements of deportation Asino and Jaja, Novosibirsk Province, Siberia, Soviet Russia, in the Summer of 1941. One hundred fourteen cases were confirmed by serologic and/or allergic tests and 115 out of the 121 cases were the ulceroglandular type. The epidemic was transmitted by insects, probably mosquitoes, and the source of the infection was water rats.

A striking feature of this uncommon epidemic was an early and transient involvement of the central nervous system, manifested in signs of meningism, serous meningitis, and at times meningo-encephalitis. Almost one-half of the total of the hospitalised cases (twenty-eight of fifty-eight) showed a characteristic and unusual tetrad: meningism, high fever, a primary tularemic focus in form of a small papule or pustule on the leg or foot, and painful regional femoral or inguinal lymphadenopathy.

The involvement of the central nervous system usually occurred in the early days of the disease and lasted for two to ten days with an average of about five days. In most of the cases the central nervous system involvement regressed completely and did not change the outlook. It consisted mostly in transient meningism with a stiff neck, positive Kernig and Brudzinski signs, and severe headache relieved by lumbar puncture. In most cases of meningism the spinal tap showed an increased pressure of the cerebrospinal fluid without marked inflammatory signs. In several instances the long persistence of meningeal symptoms, as well as the increased amount of protein and slight increase in cells in the clear cerebrospinal fluid which flowed under high pressure, suggested existence of a serous meningitis. In four cases clinical signs of meningo-encephalitis were observed, one of which resulted in death.

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PHYSICAL MEDICINE IN NEUROMUSCULAR AFFECTIONS

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PHYSICAL therapeutic agents play an indispensable role in the diagnosis and treatment of many affections of the neuromuscular system. Controversial and moot points of their physiological effects have been cleared up in recent years and improved methods have been added. The trend towards "total rehabilitation" has broadened the working basis of physical medicine, integrating the use of occupational therapy and reconditioning. Only three of the most frequent types of neuromuscular disorders can be considered in the limited scope of this discussion: peripheral nerve lesions, infantile paralysis and hemiplegia.

PERIPHERAL NERVE INJURIES

Injuries of peripheral nerves result in immediate, complete or in complete paralysis of the muscles supplied by the injured nerve. The nerve will undergo minor disturbances of function, partial or full degeneration—distal from the site of the lesion, if there is separation of the muscles from their trophic center, they become weakened or fully paralyzed and suffer from lack of nutrition and exercise. They also may suffer from overstretching by the unaffected antagonists if allowed to remain in a faulty position. The other soft parts, as well as bones, joints and tendons suffer from lack of nutrition and may develop adhesions, faulty positions, trophic ulcers and other pathological conditions. Hence the necessity for appropriate physical measures, in connection with indicated surgery, to counteract these conditions.

In many cases it is clinically impossible to ascertain at first whether the vitality of the nerve axons has been sufficiently damaged or broken to cause progressive nerve degeneration below the site of the injury. Electrodiagnosis enables a differentiation to be made after ten days or so between more serious lesions leading to a reaction of degeneration and minor injuries which recover spontaneously. There is no immediate electrical test available which will ascertain whether or not there is a separation of nerve fibers to such an extent that spontaneous regrowth is impossible. However, even immediate nerve suture does not prevent subsequent nerve degeneration. As long as the ends of an injured nerve remain in close anatomic approximation, regeneration of the nerves takes place automatically by the descent of new axis cylinders from the intact central end at a rate of from 1 to 4 mm. a day, but restoration of nerve function occurs much later. The anatomic

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regeneration of the nerve can be assisted only indirectly by improvement of the circulation and nutrition.

Physical therapy holds the first place in the treatment of peripheral nerve lesions; operative procedures will not serve to restore function but will only make it possible for the nerves to regenerate. Physical therapy must be initiated promptly to the end that when the nerve regenerates it will activate a mechanism capable of adequate movement.

Regarding *electrodiagnosis*, the classic faradic-galvanic test is still the simplest and most convenient test for qualitative study. Positive response to a tetanizing stimulus, as furnished by the faradic coil or by the 60 cycle alternating current (which in number of short stimuli is essentially like the faradic current), offers an unmistakable visual observation that the nerve still contains an appreciable number of undegenerated fibers. The response to the impulse ("make") of the galvanic current is a brisk contraction in a normal and a sluggish muscle, a more sustained one in a denervated muscle. Repeated investigation about the validity of the formerly much emphasized change of the "polar formula"—increased response to the positive pole in degeneration, instead of more response to the negative pole under normal conditions—has shown that the response may become equal but never truly reversed, hence it may be very well disregarded. Improving response to galvanic stimulation may indicate recovery before voluntary power begins to return.

For quantitative study of excitability and for discovering signs of recovery, a number of newer tests have been developed, most of which however demand more complicated equipment and laborious plotting as well as considerable experience and theoretic knowledge. Among these tests are, in order of their introduction: condenser testing, chronaximetry, progressive currents, strength-duration excitability curves, electromyography and finally the determination of skin resistance. Each of these methods has advantages in certain circumstances. In addition it must be remembered that electric stimulation of exposed nerve at operation might prove a nerve responsive which had not been shown so when tested through the intact skin.

Treatment.—For treatment of nerve injuries by physical agents, the aims are to improve circulation by heating, diminish wasting and fibrosis by electric stimulation and maintain mobility of joints by active and passive exercise and massage. Among the principal physical measures are splinting, heating, electric stimulation, exercise and occupational therapy.

Splinting is a measure of primary importance in maintaining anatomical rest and relaxation of paralyzed muscles and protecting them against constant pull of their antagonists; on the other hand splinting must not be too rigid or kept up indefinitely or it will cause atrophy of nonparalyzed muscles, loss of mobility of joints, adherence of

tendon structures. Splints must be simple, light, easily applied and removed; recently splints have been constructed which mobilize the paralyzed parts rather than hold them rigidly in one position from the time of injury.¹ Splints should be removed several times a day for the application of physical therapy.

Heating is an important introductory measure to all forms of exercise and for improving circulation, local nutrition per se. Because of disturbed sensation all forms of heat must be employed with great caution and especially so when scar lesions are present, adding a defect of collateral circulation which acts as an impediment to dissipation of excess heat. The relatively safest and most desirable measure for extremities is the whirlpool bath, applied daily at a temperature below 105° F. for at least one-half hour. Luminous heat is fairly safe, while diathermy is generally contraindicated because the control of a safe dosage depends as much on sensory control as on the meter reading.

Electrical stimulation is an essential measure in treatment of nerve injuries. Clinical observation has established its value for quite some time and recent experimental evidence has amply corroborated it. The indifferent results obtained in the very few earlier studies have been due to inefficient and infrequent stimulation. Among recent positive laboratory observations are those of Wehrmacher and associates² showing that atrophy of muscle following denervation can be appreciably retarded by electrical stimulation; stimulation developing the greatest amount of tension results in conservation of a large mass of functional tissue. Jackson and his co-workers³ report that daily treatment in a number of controlled cases of ulnar paralysis almost entirely prevented wasting except during the week immediately after denervation; even during this period it decreased the rate of wasting. It therefore seems well established that in peripheral nerve injuries, electricity is valuable in combating atrophy, lessening fibrosis and maintaining elasticity of paralyzed muscles. It is important to begin this treatment as soon as possible and continue it until voluntary power returns. The number of stimuli may be small and duration of treatments brief, especially if treatment with rest periods between can be repeated several times daily.

As to the most efficient type of current, Wehrmacher's group recommended shocks by a strong induction (faradic) coil while the Northwestern University group found⁴ the 25 cycle alternating current most effective in eliciting maximum contraction with a minimum of current strength. Such a current is not furnished by most of the older types of low frequency apparatus, but some of the recent apparatus offers a low tension current which is fully regulable not only as to strength but also as to rate of frequency and duration of its peak strength. I have for many years used satisfactorily the surging galvanic or slow sinusoidal current, utilizing its impulses of slow increase and long duration. In cases of partial reaction of degeneration with only slight quantitative

electrical changes, stimulation by a current of a surging faradic type may be employed.

Corresponding with the progress of anatomic regeneration of the nerve, the atrophy of the muscles gradually disappears and their response improves. After many weeks or months a time comes when there is the first indication of return of voluntary motion in one or more of the paralyzed muscles. This is proof that the nerve trunk and the motor end plate have regenerated.

Massage as an aid to circulation and for the improvement of nutrition is a useful part of the routine physical treatment. So far as the prevention of muscle atrophy is concerned, massage is only of slight benefit. Massage should always be preceded by a thermal measure and administered to the well supported and completely relaxed extremity. It should begin with rhythmic superficial stroking and continue with gentle kneading of the skin and of any contracted muscles, with friction over ankylosed joints. The average massage treatment should take from ten to twenty minutes.

Passive movement in the form of *manipulation* of joints and stretching of muscles in peripheral nerve injuries is of even greater importance than routine massage; however, it is best carried on in conjunction with massage. Contractures and adhesion of joints, muscles and tendons are due to shrinkage of newly formed connective tissue. Introductory heating and gentle manipulation serve to stretch it and slowly free it. Such manipulation will also enable normal joints to keep active and increases the range of motion in those which already have become impaired.

Active exercise and reeducation of muscle function comprise the most important part of the treatment even before active power to the affected muscles has begun to return. Since a nerve injury causes loss of function of an extremity far beyond the structures that are actually involved—because the normal use of the extremity has been lost—the early use of suitable exercises will reduce much of the unnecessary waste of muscles and stiffening of joints; it also counteracts the tendency to trick movements and helps to prevent contractions. Hence, even in the early treatment of nerve injuries, exercises are indicated for maintaining the function of muscles not directly affected and also for maintaining the full range of joint motion. When signs of active power appear in the affected muscles, the simplest possible active exercises with these muscles are begun, at first with as much assistance by the technician as required. Movements on a powdered board or under water will greatly aid in overcoming gravity and in allowing gradual redevelopment of function in the very weak muscles. Exercises must be carried on in all instances with the parts properly supported and previously warmed. It is very rarely necessary to use artificial exercise apparatus for this purpose, but use can be made of the various types of simple active exercise devices with weights and pulleys.

Recent investigations have shown that the dangers of overtiring muscles have been overemphasized in the past. Marked fatigue through artificial stimulation or forced exercise was not found to have a delaying effect on recovery.

Occupational therapy in the reactivation of nerve injuries has been extensively used in recent years from the very beginning for sustaining the patient's morale during the long convalescence as well as offering a variety of graded exercises by proper selection of craft or shop work. There is an increasing tendency to employ occupational therapy side by side with exercise therapy, skilled occupational therapists are able to direct activities not only of a coordinated type but also properly adjusted to the affected part's capacity. Such activity engages the patient's interest more than monotonous repetition of stereotyped exercises.

Bell's Palsy.—Bell's palsy or rheumatic facial paralysis is perhaps the most frequently encountered form of peripheral nerve lesion. Physical medicine should play a predominant role in its management, both for prognosis and for treatment. Since the majority of cases occur in women, an early prognosis by simple electrodiagnosis is most desirable, instead of depending on clinical impression only. Light cases responding with a normal electric reaction usually show full recovery in three to six weeks, and with a minimum of treatment, moderately severe cases with partial degeneration may take three to five months for recovery, while severe cases with full degeneration may last up to one year and may lack complete recovery.

The advice that no treatment except rest should be given until the ten day period for the possible development of degenerative nerve changes has passed, seems illogical in the view of the present day conception that chilling causes a deep-seated congestion and edema resulting in pressure on the facial nerve. The immediate use of 'decongestive' physical treatment by penetrating heat or galvanism followed by suitable electric stimulation is fully indicated and has proved clinically more effective than taking the chance that a few patients may recover without any treatment. Sagging of the paralyzed side of the face should be prevented by elastic strapping, reeducational exercises should be used for helping to maintain muscle tone. Massage is of comparatively little direct value in these cases except for giving a feeling of comfort. In severe cases late contractures or "tic" movements may occur; they are due to hyperirritability and their appearance calls for strictly sedative measures, such as diathermy and gentle stroking massage.

Other Conditions.—Among other frequent forms of peripheral paralysis due to simple compression are musculospiral and peroneal paralysis, also axillary (circumflex) nerve paralysis—this latter is due to shoulder injuries, especially dislocations. Recovery in these cases is usually fairly prompt under the procedures outlined. Brachial plexus

injuries belong among the most trying forms of lesions and may require consistent treatment extending from one to two years. Close cooperation with the neurosurgeon is of course essential in all cases.

INFANTILE PARALYSIS

The conception of physical treatment of infantile paralysis has undergone considerable changes during the past few years. This is undoubtedly due to widespread study and reevaluation of treatment as well as to early and intelligently applied physical treatment, following Sister Kenny's observations.

The physiopathology of poliomyelitis consists of inflammatory changes in the spinal cord, inhibiting or gradually destroying the function of the motor cells in the anterior horns. Some of the earlier damage is apparently reversible,⁶ which accounts for the often remarkable recovery from extensive paralysis. The modern concept of cord function is much more complex than earlier taught. The lesion of internuncial neurons⁷ in the gray matter of the spinal cord is held responsible for widespread muscle spasm, the existence of which was very little recognized formerly and the neglect of which according to our present knowledge is responsible for much of the preventable damage in the wake of poliomyelitis. It has also been established that poliomyelitis with lesions restricted to the spinal cord is rare.

Treatment.—The first objective of treatment in all cases of poliomyelitis must be the saving of the patient's life,⁸ and especially the prevention of respiratory failure; for the spinal type of the disease an *artificial respirator* or iron lung serves to take over the work done by the muscles of respiration when they have no reserve power and tire rapidly; in the bulbar type the respirator will be of no value; in respiratory difficulties due to "spasm" of the muscles of inspiration on the other hand, the skillful application of hot packs to the chest may often relieve "spasm" without recourse to the respirator.

The generally established orthopedic management of acute cases consisted until recently chiefly of strict *immobilization* of the affected parts, in order to lessen tenderness and prevent contractures. Although it was observed that during this period muscles undergo rapid wasting and there is extensive loss of function, physical treatment toward restoration of function was as a rule not started until all tenderness disappeared. The newer observations showed that early physical treatment consisting of *moist heat* and *careful passive motion* relieves pain and releases tightness, while the preservation of normal body mechanics by proper position and by the early restoration of mobility in muscles and joints acts as a preventative of deformities due to faulty alignment. The generally followed method of applying heat consists of the use of hot "fomentations," for which Sister Kenny has established a rather rigid technic; it is probable that a less time-consuming and less expensive form of heat application may be developed, as indicated

by the report that hot pool treatment⁹ may be more efficacious and simpler in relieving muscle "spasms" and pain.

The early training of patients to use every available muscle fiber with the greatest possible efficiency is the crux of the newer treatment of infantile paralysis; it requires the painstaking service of a highly skilled and intelligent physical therapist. Carefully graduated activity serves to increase muscle strength and functional activity gradually. Regular charting according to standard methods keeps track of the progress. The achievement of maximum results may require but a few weeks in mild cases and several years in severe cases. It is generally conceded that the early use of physical therapy prevents much of the muscle contracture and dysfunction following the older method of rigid immobilization, resulting in less late deformity and greater functional ability. But there has been no acceptable proof offered anywhere that the incidence of residual paralysis has been changed to any extent; in other words the muscular units which have received permanent primary injury from the activity of the virus remain paralyzed under any form of presently known treatment.

As an alternate or supplement to muscle reeducation in the early stages *underwater exercise* in therapeutic pools offers a desirable means to combine heat treatment with graduated muscle exercise; due to the buoyancy in water weak muscles unable to act against gravity outside the water can be exercised under water. When underwater exercises are not readily available one may employ the simple procedure of moving a limb on a smooth, well powdered board in such a way as to eliminate the pull of gravity during exercise of an involved muscle; or the extremity may be carried in a sling attached to an overhead frame, allowing the extremity to swing through the arc in which the involved muscle contracts.

There exists a definite role for *electric stimulation* in infantile paralysis in advanced cases, as reported by me many years ago;¹⁰ its rationale is similar to that outlined under peripheral nerve injuries. Lewin¹¹ holds that electricity may improve the local condition in poliomyelitis and induce contractions in muscles otherwise inaccessible; in trained hands, used as a simple muscle tonic, electricity should be helpful. There is an increasing amount of clinical and experimental evidence that electrical stimulation is of definite aid in recovery of paralysis. Hansson¹² states: "I should not be surprised to see return of electrical stimulation to the weakened muscles, making the treatment of poliomyelitis similar to the care of traumatic nerve lesions." According to clinical experience the most amenable cases for electric stimulation are those in which only a few muscles are affected. Electric stimulation must always go hand in hand with muscle reeducation, just as in peripheral nerve injuries.

Even under present day advanced methods of physical therapy there will occur cases in which *orthopedic surgical procedures* are necessary to

enable safe and practical locomotion; newer methods of light and elastic prosthesis construction will do much to minimize the appearance of the unavoidable muscle atrophy and deformities and restore the contour of an atrophied leg.¹⁸

HEMIPLEGIA

Hemiplegia is usually the result of a primary disease of the vascular system, either an arterial obstruction or hemorrhage. An apoplectic stroke usually comes on suddenly and is accompanied by focal signs which reflect the loss of function of the affected parts of the brain. As a result there remains a distressing train of symptoms that may include disturbances of speech, of gait, of the movements of the arm and hand and often mental lassitude, dizziness and loss of memory. Most of these symptoms tend to disappear spontaneously. The extreme limit of spontaneous restitution ends between the ninth to twelfth month.

Treatment.—Physical therapy has a definite place in this condition for the early rehabilitation and reeducation of affected extremities, hastening convalescence and restitution and contributing to mental ease. The physical therapist as a rule does not see many cases of hemiplegia in the acute stage except in general hospitals. Early treatment gives the patient a better chance of recovery; the final result depends on the extent of the subsequent recovery of the affected motor pathways. In the first week of hemiplegia, while the patient may be still in bed, the application of *radiant heat* to the affected arm or leg will comfort and tend to relax the paralyzed limbs. Faulty positions of the joints with subsequent contractures are prevented by simple *splinting* and frequent changes in the position. *Passive movements* should be performed early and be accompanied by movements of *massage*. As soon as some muscular power has returned, the patient should be encouraged to do *active exercise*, thus counteracting the tendency to contracture.

In the subacute stage, when the earlier flaccid paralysis gradually changes into spasticity, gentle *stroking massage* and *muscle reeducation* are of paramount importance. The application of some form of heating, usually radiant, should always precede the physical treatment. *Underwater exercises* in a tank or pool have been successfully employed in recent years to relax the spastic muscles and to encourage voluntary use. Hydromassage of the affected limbs by a *whirlpool bath* is an efficient combination of heating and massage, and most patients take well to it; keeping the temperature of the water just comfortably warm around 100° F. will prevent overheating of which some heat-sensitive patients are apt to complain.

Electrical stimulation for muscle exercise has a definite but minor role in hemiplegia. Its value is doubted by those who feel that spastic muscles are only too prone to respond to any stimulus; it is recommended, therefore, that electrical treatment should be applied to the

opposing normal muscles in order to prevent their overstretching. Most observers agree that this is rather far-fetched and that gentle electric exercise tends to relax and contract the spastic muscles and serves to maintain nutrition during the process of recovery. Its chief value lies perhaps in the encouragement it renders when patients see their seemingly helpless limbs move; they are more willing to follow through with efforts for active exercise. It can be applied with the standard monopolar technic or by the group stimulation of muscles with the hand and one foot in separate basins with salt water. Occupational therapy when appropriately employed serves the same double purpose; it serves as a means for suitable exercise and also keeps up the patient's morale.

There are a few special treatment methods available for the relief of certain symptoms. *Cerebral galvanism* as well as *diathermy* has been employed by a number of clinicians, including myself, in patients complaining of headache, dizziness or simple mental lassitude not due to continued hypertension. Application is simple and perfectly safe in skilled hands; it can be done daily at first and later every other day. For paresthesias and other painful sensations the *monoterminal high-frequency* (Oudin) current from a long wave spark gap machine may be applied advantageously. The glass condenser electrode is rapidly moved over the well powdered skin and is held in close contact to the parts. The current is regulated so as to cause just a gentle pricking and mild surface warming.

Alongside the general medical care, the relief of symptoms and gradual muscle reeducation, nowadays the "*total rehabilitation*" aspects of the condition must also receive careful consideration. Patients must first be made safely ambulatory, and be taught self-care so as to cope with the demands of daily living. Unfortunately, the recovery of motor ability does not correlate well with the return of functional capacity in the hemiplegic patient. A recent study¹⁴ has shown that an organized program of rehabilitation and retraining definitely increases the recovery of functional capacity. It is hoped that in accordance with the recent trend for a third phase of medicine hemiplegic patients will have increased opportunity to partake in such programs.

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the treatment of such patients. Sometimes a team of full-time workers in cerebral palsy plus consultants or part-time workers is formed.

The team of specialists in cerebral palsy may be desirable but not always obtainable. It may be desirable especially for institutions where patients can be treated as inpatients for longer or shorter periods and where a concentration of specialists will afford possibilities for study and teaching.

In many areas where these centers are not available and where they may not be available for a long time to come, it may still be possible to give much help to the patient with cerebral palsy by a team of workers, doctors, therapists and teachers who devote only part of their activities to this purpose. It has been on this basis that the cerebral palsy clinic at the Columbia Presbyterian Medical Center has been operating.

This cerebral palsy clinic has been organized as a group clinic under the direction of Babies Hospital. It meets on the floor of the pediatric clinic. No special facilities have been created. The space allotted to the cerebral palsy clinic is used by the pediatric clinic for other purposes when the cerebral palsy clinic is not operating. Except for psychometric tests and some consultations all the work done with the children and all consultations and check-ups are made at this place. This avoids unnecessary waiting and saves the patients trips between different parts of the hospital. Children are admitted by the pediatric clinic; if necessary, they are hospitalized for a brief period for a complete work-up. A pediatrician acts as chief of clinic. He administers and directs the clinic, coordinates its activities and maintains contact between the different members of the consulting and working staff. He represents the clinic to other departments and to outside agencies. After completing the pediatric examination, the chief of clinic refers the child for further consultations if necessary.

All children, however, are routinely seen after admission and later at intervals by three consultants, as follows: (1) A neurologist who makes the complete initial neurological examination of each patient. (2) An orthopedic surgeon who advises on braces and possible operative procedure. (3) A psychiatric consultant. The children have to undergo a psychometric test, which is repeated after one year in cases of subnormal rating.

Other consultations which might be sought in individual cases are: speech specialist, x-ray, ear, nose and throat specialist, ophthalmologist, pediatric surgeon, dentist and orthodontist. The family situation is investigated by a social worker and, if necessary, by a psychiatric social worker who helps advise the parents. When major difficulties arise, both parents and children are advised and treated by a *consulting psychiatrist*.

After these examinations have been made, the children are referred for physical and occupational therapy. Muscle tests are made by the

attending doctors and treatment plans, including exercises and occupational therapy, are outlined. The treatment plans are tentative at first because the admission of new patients for treatment is always made on a trial basis. This is necessary because outside circumstances which cannot be foreseen at the beginning may interfere with successful treatment. This outside interference may be lack of cooperation by the parents, inability to bring the child in for regular treatments, or a difficult family situation. Furthermore, the trial period of treatment of one to two months makes it possible to evaluate whether the child is trainable or not and an appraisal of his mental limitations and capacity can be made. This in addition to the psychometric test permits better evaluation of the patient.

The physical therapy given to the patients consists of exercises. The exercises are prescribed according to the needs and merits of the case. No special "exercise system" is adhered to. When the muscle tests are made, the muscles requiring treatments are charted. In the muscle chart, weaknesses, spasticity and contractures are noted and graded. The exercises are directed mainly to the most obvious and most crippling deficiencies. Minor difficulties are at first purposely disregarded. This makes it possible to give more intensive exercises to the parts being treated and thereby increase the effect of these exercises.

In athetosis "joint motion charts" are made. "Balance graphs" or "coordination graphs" may supplement the muscle tests in ataxias.

A tongue motion test is made if speech difficulties are present and a "speech chart" noting the deficient letters and simple combinations is started. If speech difficulties seem to be caused by other than muscle dysfunction, the child is referred to the speech consultant for evaluation and, if necessary, training.

At the same time that the exercise program is started the occupational therapy program is begun. It usually starts with the "handedness test" in order to avoid interference with the "handedness" of the child. This is especially important when speech difficulties or a history of seizures is present.

As soon as possible the elements of self-help (feeding and clothing) are taught. An "achievement chart" guides the beginning of both the occupational and physical therapy programs.

While an attempt is made to increase the "achievements" as quickly as possible, occupational therapy is used not only to teach new skills but to teach the correct, well performed accomplishment of skills already familiar to the child. The stressing of "form" over actual accomplishment prepares the way for further increases in planned activities. In other words, while some occupational therapy measures and most of the physical therapy exercises aim at "improving the motor abilities that the patient has left," some therapy aims only at "making him get along with what he has left."

Some mechanical aids such as "skis," "walkers," standing tables, relaxation chairs, etc. are used in this teaching process.

Since the clinic is an outpatient clinic only, run on a half day basis six times a week, great stress is laid upon the cooperation of the *parents*. Not only are the parents instructed to carry out the exercises at home but an attempt is made to guide them with occupational therapy. Child behavior is discussed with them. The parents have formed a "Parents Club" which meets once a month. Common problems are discussed and speakers are asked by the parents group to talk on points of interest. These meetings have been helpful, too, in promoting understanding and contact between parents and doctors as well as therapists.

Twice a month a conference is held under the direction of the chief of the cerebral palsy clinic. At this conference all the therapists, social workers and the medical staff interested in the treatment of the children are present. At every conference special problems are brought up and a number of cases, progress reports and complications are discussed by the group. Any measure of major importance to the child is discussed at this conference clinic before being put into effect; institutionalization, the discontinuance of treatment and the like are always discussed before being acted upon. It is in these conferences that the possibilities of further rehabilitation are discussed and planned. This includes schooling. Later vocational planning will be attempted. In order to record and follow the different decisions of this conference a "correlation sheet" is used. This printed form gives a list of all the different specialists and agencies the patient might be referred to and it enumerates the more frequent suggestions made. The results of consultations are recorded on this form as well as decisions made or requests for more consultations, special treatments, assistance from social agencies, etc.

A social agency provides *transportation* for some of the children who otherwise could not be taken to the clinic. This same agency provides home treatment for other cases where necessary. These home cases come in for joint clinics held at regular intervals with the representatives of the social agency.

This type of group clinic offers real possibilities for communities where a special cerebral palsy center cannot be organized. It has proved of great educational value for all the members of the group who have been able to learn from one another and to familiarize themselves with the whole problem of cerebral palsy instead of being restricted to one phase of it.

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THE DIFFERENTIAL DIAGNOSIS OF COMA

LESTER A. MOUNT, M.D.*

COMA is a state of unconsciousness from which the patient cannot be aroused. The etiology of coma encompasses almost the entire field of medicine. The treatment, whether medical or surgical, depends upon the cause. The acute causes of coma offer the most difficulty in differential diagnosis. Coma may be the terminal stage of almost any disorder.

Diseases directly involving the brain:

- | | |
|---------------------------------|--|
| 1. Brain injury | 10. Encephallitis |
| 2. Subdural hematoma, acute | Epidemic |
| 3. Subdural hematoma, chronic | Secondary to exanthematous diseases |
| 4. Epidural hematoma | 11. Brain abscess |
| 5. Cerebral hemorrhage | 12. Meningitis |
| 6. Cerebral thrombosis | Meningococcic, tuberculous, influenza, other |
| 7. Thrombosis of venous sinuses | 13. Brain tumor |
| 8. Cerebral embolism | 14. Postconvulsive coma |
| 9. Subarachnoid hemorrhage | |

Diseases not primarily involving the brain:

- | | |
|--|--|
| 1. Severe fevers | 7. Callosal disease |
| 2. Metabolic disease | 8. Shock |
| Diabetic coma, Addison's disease, myxedema | Due to injury |
| 3. Uremia | Due to hemorrhage from intestinal tract, lungs, ruptured tubal pregnancy, ruptured aneurysm, other |
| 4. Stokes Adams syndrome | 9. Excessive temperature |
| 5. Coma due to poisoning | Heat stroke, heat exhaustion, freezing |
| Alcohol, bromides, barbiturates, lead, morphine, other | 10. Hysterical coma |
| 6. Coma due to gases | 11. Simple fainting |
| Carbon monoxide, carbon dioxide, hydrogen sulfide (sewer gas), other | |

SYMPTOMS AND SIGNS

History.—The history will most often give a clue as to the etiology. When there is a history of trauma, diabetes, renal disease, epilepsy,

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high blood pressure, taking of drugs or exposure to gases, the diagnosis is not difficult. Frequently the history is entirely absent.

Physical Examination.—The examination, too, may indicate the causative factor. The sex and age of the patient are not especially helpful although in young patients trauma, meningitis or epilepsy are the more likely causes. The points in the examination of especial help in the differential diagnosis are the following:

Pulse.—A slow, full, bounding pulse suggests increased intracranial pressure, the result of trauma, hemorrhage or tumor. A full, bounding pulse may also be present in patients with hypertension or nephrogenic disease. A slow pulse is found in patients whose coma is the result of freezing, morphine poisoning, brain tumor or other expanding lesion and Stokes-Adams syndrome. An irregular pulse directs attention to the heart, and if there is evidence of auricular fibrillation, valvular heart disease or bacterial endocarditis, cerebral embolism is the probable cause of coma.

Respiration.—Loud and stertorous breathing occurs in patients with apoplexy; Kussmaul breathing, in patients with diabetic acidosis; and rapid breathing, in patients with fevers or infectious diseases. Slow respiration suggests early increased intracranial pressure, morphine poisoning or freezing.

Blood Pressure.—An elevated blood pressure hints of uremia or cerebral hemorrhage, but it must be remembered that the blood pressure is often lower immediately after a cerebral hemorrhage than it was before the hemorrhage.

Temperature.—Most patients in coma have an elevated temperature, but the temperature is below normal in patients with morphine poisoning or freezing.

General Appearance.—The cherry red color of the patient with carbon monoxide poisoning is characteristic. Patients in diabetic acidosis often have a red face early and later a pale and cyanotic one. A flushed face is found in the alcoholic and a pale yellowish one in the uremic. A cold, clammy skin suggests hyperinsulinism or morphine poisoning. Pale brown pigmentation of skin and buccal mucous membranes intimate Addison's disease. A powdery feeling of the skin (uremic frost) may be found in uremic patients. Petechiae in skin, nail beds, conjunctivae, or mucous membranes suggest cerebral embolism as the cause of coma. Edema of the ankles hints of uremia or cerebral embolism. The findings of contusions or lacerations of the scalp, blood or cerebrospinal fluid in the nose or ears favors the diagnosis of cerebral trauma: concussion, contusion, laceration or hemorrhage in the brain. A depressed fracture of the skull is sometimes palpable and the presence of brain tissue in a laceration of the scalp is diagnostic of a compound depressed fracture of the skull.

Odor.—The odor of whiskey on the breath denotes alcoholism, but there may be an associated serious head injury. The sweet odor

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there may be no lucid interval. A fracture across the middle meningeal artery in the frontotemporal region and a shift of the pineal to the opposite side are valuable supportive evidences.

Acute Subdural Hematoma.—The diagnosis of acute subdural hematoma should be kept in mind in all patients who have had a head injury or in alcoholics without such a history. There are no set diagnostic criteria. Headache, mental disturbances, dilatation of one pupil, fluctuation in the state of consciousness and focal or scattered neurologic findings are suggestive of a subdural hematoma, and a shift of the pineal is suppositive evidence. Any patient with a head injury who is not doing well should have bilateral trephination to rule out an acute subdural hematoma.

Chronic Subdural Hematoma.—The history of head injury (or alcoholism), followed weeks or months later by headaches, nausea, vomiting and disturbances in vision, is strongly suggestive of a chronic subdural hematoma. The headaches may be intermittent but gradually become more severe. The visual disturbances usually consist of blurring of vision. Mental symptoms appear or become more marked as the headaches increase. These vary from impairment of memory to complete disorientation. Weakness in the extremities on one or perhaps both sides may appear but when it is unilateral it is not necessarily localizing as the paresis develops almost as frequently on the ipsilateral side as it does on the contralateral side. Later the patient becomes unconscious. The state of unconsciousness may vary widely within a few minutes. The patient may be comatose and yet within a few minutes he may be reading a newspaper. This variation is characteristic of a chronic subdural hematoma. The examination shows papilledema and scattered neurologic findings. In those patients with a calcified pineal gland the x-ray reveals a shift of the gland if the hematoma is unilateral, but if the hematoma is bilateral the pineal gland may not be displaced. Spinal puncture shows yellow fluid under increased pressure and containing an increased total protein. Bilateral trephination of the skull verifies the diagnosis.

Differential Diagnosis of Cerebral Hemorrhage, Cerebral Thrombosis and Cerebral Embolism.—The differential diagnosis of these conditions may be difficult. High blood pressure favors cerebral hemorrhage. Cerebral hemorrhage occurs in a younger age group than thrombosis. The onset in hemorrhage is sudden and coma is immediately preceded by headache in some cases. Blood in the cerebrospinal fluid supports the diagnosis. High blood pressure need not indicate hemorrhage as it is often associated with disease of the blood vessels. Disease of the blood vessels, whether arteriosclerotic or syphilitic, promotes thrombosis. Thrombosis occurs in older patients, unless syphilis is the cause, and frequently takes place during sleep. It more often occurs in patients with heart disease and low blood pressure. Premonitory symptoms such as numbness, transitory weakness or temporary speech

LABORATORY EXAMINATION

Laboratory tests lend further aid in making the diagnosis. Sugar, diacetic acid, and acetone in the urine and elevated blood sugar obviously make a diagnosis of diabetes. A low blood sugar is found in hyperinsulinism, and Addison's disease. Uremia is characterized by the elevated blood urea and nonprotein nitrogen, but both may be elevated in Addison's disease also. Elevated cerebrospinal fluid pressure may be present in patients having uremia or high blood pressure, but is more characteristically elevated in patients having brain tumor, brain abscess or intracranial hemorrhage. Yellow fluid or blood in fluid is indicative of hemorrhage. Cloudy fluid containing an elevation in white blood cells is present in patients having meningitis or brain abscess, and in patients having hemorrhage which spread into the subarachnoid space twenty-four hours previously. A moderate increase in the cell count is found in encephalitis. An elevation in sugar in the cerebrospinal fluid is strongly suggestive of encephalitis when the blood sugar is normal. The Wassermann reaction in the cerebrospinal fluid is strongly positive and the gold sol test shows a paretic curve in patients having general paresis. Gastric analysis will show the cause in patients who have taken drugs only a short time previously. The electrocardiogram will confirm the diagnosis of Stokes-Adams syndrome.

X-ray Examination.—The x-rays of the skull show atrophy of the sella turcica in patients having increased intracranial pressure of three months' duration. The pineal gland may be displaced when an expanding lesion is present. This shift can be demonstrated in the skull films when the pineal is calcified. Calcified tumors are demonstrable by x-ray. There may be erosion of the skull by some tumors, thickening of the skull in others, and increased vascularity in still others. X-ray evidence of a carcinoma in the lungs is good presumptive evidence of metastatic carcinoma of the brain.

CLINICAL SYNDROMES WHICH REQUIRE FURTHER CLARIFICATION IN THE DIFFERENTIAL DIAGNOSIS OF COMA

Cerebral Injury.—The history of a cranial injury suggests cerebral injury as the cause of coma. Whether the patient has a cerebral concussion, cerebral contusion or cerebral laceration and hemorrhage depends upon the nature and extent of the trauma. Fracture of the skull is important only in its relationship to the cerebral injury.

It is important to consider the presence of an epidural or a subdural hematoma because operative therapy is indicated when either of these is present.

Epidural Hemorrhage.—The typical history for an epidural hemorrhage is that of injury with unconsciousness, a lucid interval, increasing hemiparesis, loss of consciousness, and a dilated pupil. However,

there may be no lucid interval. A fracture across the middle meningeal artery in the frontotemporal region and a shift of the pineal to the opposite side are valuable supportive evidences.

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disturbances sometimes occur before the onset of hemiparesis and coma. Coma is more frequently absent in cerebral thrombosis than it is in cerebral hemorrhage or cerebral embolism. Embolism most frequently occurs in younger patients and more often involves the left side of the brain. The diagnosis can be made only when a source of the embolus, such as endocarditis, is evident.

Coma Due to Acute Encephalitis.—Coma due to acute encephalitis is readily recognized during epidemics, but may be missed at other times. Encephalitis usually starts with generalized malaise, upper respiratory infection, headache and low grade fever. Later diplopia, drowsiness, delirium and coma appear. Neurologic examination shows variable signs and it may be difficult to differentiate encephalitis from brain tumor or brain abscess. The cerebrospinal fluid pressure is usually increased in all three conditions but more so in the latter two. The total protein in the cerebrospinal fluid may be increased in all three conditions but is less high in encephalitis and abscess. The white cells may be increased in all three conditions. Early there is a preponderance of polymorphonuclear leukocytes in both encephalitis and abscess but later there are more lymphocytes. The elevation of the sugar in the cerebrospinal fluid is strongly in favor of encephalitis.

Coma Due to Intracranial Tumor.—Coma due to intracranial tumor is preceded by headache, nausea, vomiting and visual disturbances. Other symptoms depend upon the area of the brain involved but the course is progressive. This progressive course aids greatly in the differentiation of tumor from cerebral hemorrhage, embolism or thrombosis. The onset in embolism is sudden and is maximal immediately. The course in thrombosis may be progressive but more rapidly so than in tumor and more apt to be steplike in its progression. The examination gives no aid in differentiation except for the presence of papilledema in tumor, arteriosclerosis or syphilis in thrombosis, and petechiae, auricular fibrillation or valvular heart disease in embolism. In x-rays of the skull atrophy of sella turcica, calcification in the cranial cavity, displaced pineal gland, hyperostosis, or increased vascularity distinguish tumor, and calcification in the walls of the internal carotid artery suggests thrombosis. Increased pressure of cerebrospinal fluid favors tumor; a positive Wassermann or positive colloidal gold, thrombosis; and positive electrocardiographic findings favor embolism.

Cerebral Abscess.—The symptoms and signs of cerebral abscess are very similar to those of cerebral tumor, but the course is usually more rapid. Symptoms and signs of sinusitis, otitis media, mastoiditis, lung abscess or bronchiectasis intimate the presence of brain abscess. Fever and leukocytosis also are in favor of abscess although both may be absent, even with large abscesses. Increased cells in the cerebrospinal fluid aid in the further differentiation from brain tumor.

Subarachnoid Hemorrhage.—Subarachnoid hemorrhage is usually the result of rupture of an aneurysm. When hemorrhage is great consciousness is lost. The history in such cases is that of sudden onset

of severe headaches, usually in the back of the head, loss of consciousness, and on regaining consciousness the patient has a stiff neck and a positive Kernig's sign. Spinal tap shows bloody fluid. The site of the bleeding point may not be evident but the most frequent source is the circle of Willis. Similarly, the most common site for an intracranial aneurysm is the internal carotid artery. When the aneurysm involves this vessel the patient may develop unilateral ptosis, a dilated pupil which does not react to light, and an eyeball which is rotated outward. He may develop contralateral weakness with hyperactive reflexes and a positive Babinski reflex or its confirmatories.

Thromboses of the Venous Sinuses.—Thromboses of the venous sinuses are infrequent causes of coma. The etiology is infection, trauma or pathological vascular states. Cavernous sinus thrombosis is secondary to infection of the face, nasal sinuses, throat or ears. Coma is much more common when thrombosis is secondary to infection of the face, as the course is more fulminating. The sequence of events is pain about the eye, fever of septic type, orbital congestion, swelling of the eyelids, conjunctivae, forehead, nose and cheek, chemosis, ptosis, exophthalmos, limitation in movement of the eyeball, dilated or constricted pupil, normal or impaired vision and papilledema. The first division of the fifth cranial nerve may be involved with pain early and with impaired sensation later. These findings may be bilateral. The course is rapid and delirium or coma are frequent.

Thrombosis of the Superior Sagittal Sinus.—Thrombosis of the superior sagittal sinus is characterized by *caput medusae*, i.e., fullness and tortuosity of the veins of the scalp, forehead and eyelids. Severe headaches, vomiting, papilledema and mental symptoms are usual. Convulsions, hemiplegia or paraplegia, rigidity and coma may occur.

Postconvulsive Coma.—The postconvulsive coma is undistinguished many times from coma due to other causes. The patient is relaxed, the pulse is rapid and full, breathing is stertorous and the face is congested. If there is foam at the mouth, if the tongue has been bitten or if there are scars on the tongue secondary to previous seizures, the diagnosis is less difficult. Incontinence of urine or feces is supportive evidence. Knowledge that the patient has recently suffered from a convulsion or at least is subject to them, of course makes the diagnosis secure.

Diabetic Coma.—In known diabetics coma may be the result of diabetic acidosis or hyperinsulinism. The onset of diabetic acidosis is usually slow and may take days, but is occasionally rapid. Preceding coma, the patient complains of marked thirst, frequent urination, pain in the abdomen and vomiting. He becomes restless and irritable. On examination slow, deep respiration (Kussmaul breathing) is observed. The pulse is rapid and weak, and blood pressure is usually low. The skin and tongue are dry, the eyeballs are soft, and the breath has the fruity odor of acetone. The urine shows glucose, diacetic acid and acetone, the blood carbon dioxide capacity is low and the blood sugar

is high. The last, of course, is diagnostic of diabetes, the low blood carbon dioxide of acidosis.

Hyperinsulinism.—Most of the symptoms and signs of hyperinsulinism are the opposite of those produced by diabetic acidosis. The onset in hyperinsulinism is usually sudden. Nausea and vomiting occur when protamine zinc insulin is used but rarely is found when the regular or crystalline insulin is used. Insulin reactions occur when the patient has had too little food, and hunger may be an early symptom. Headache is common, and palpitation, diplopia, apathy and confusion are usual. Muscular twitchings are frequent. The pulse is full and rapid, respirations normal and the blood pressure tends to rise. The patient is pale and the skin is moist. A tremor may be present. The deep tendon reflexes are hyperactive and a positive Babinski's reflex may be present. A low blood sugar is strongly supportive but Addison's disease and Simmonds' disease must be considered when the blood sugar is low.

Uremia.—Uremia at an advanced stage produces convulsions and coma. It is the result of deficient urinary secretion. Convulsions and coma are preceded by headache, nausea, vomiting, visual disturbances and mental disorders. The signs of uremia are a urinous odor of the breath and skin, uremic frost, elevated blood pressure, and white patches and flame-shaped hemorrhages in the retina. The urine shows albumin and casts. The diagnosis is verified by the elevation of the blood nonprotein nitrogen.

Stokes-Adams Syndrome.—Stokes-Adams syndrome usually occurs in patients with arteriosclerosis. It is the result of changing rhythm from normal to heart block, or vice versa. It also occurs in patients with heart block who suddenly increase their activities. The patient is pale at first and then becomes progressively more cyanotic. Muscular twitchings or epileptiform convulsions may occur. The auricular beats may be seen in the veins of the neck. The diagnosis can be made by an electrocardiogram.

Alcoholic Coma.—In alcoholic coma the face is flushed, the respirations slow and stertorous, the pulse rapid and full, and the temperature normal or below normal. The pupils are dilated and the breath reveals the odor of the causative agent. Alcohol in the blood over 0.1 per cent is diagnostic. Glycosuria may be present in the alcoholic but in such cases the specific gravity of the urine is low in contrast to the high specific gravity in diabetics. The alcoholic may also have had a head injury and coma cannot be dismissed as just alcoholism until such an injury is ruled out.

Acute Morphine Poisoning.—In acute morphine poisoning a stage of excitement may precede coma. Vertigo and itching are common. The skin is cold and clammy, the pulse and respirations are very slow, the temperature and blood pressure are low and the pupils pin point. Death is the result of paralysis of the respiratory center.

Coma Due to Ruptured Ectopic Pregnancy.—Coma due to ruptured ectopic pregnancy is the picture of shock with exsanguination, rapid thready pulse, low blood pressure, sighing respirations and cold extremities. Abdominal findings vary from rigidity to complete relaxation. Pelvic examination shows an enlarged uterus, boggy sausage-shaped mass in the region of the tube and fullness in the cul-de-sac.

Heat Exhaustion.—Heat exhaustion or prostration occurs most frequently in debilitated persons and chronic alcoholics who are exposed to excessive heat, especially when the humidity is high. The patient first develops a feeling of oppression, then faintness, collapse and loss of consciousness. The patient is pale and has shallow, irregular respiration, a weak rapid pulse, low blood pressure and a normal temperature; the skin is cold and clammy and the pupils are dilated. Death is the result of respiratory or cardiac failure.

Heat Stroke.—Heat stroke or heat apoplexy occurs in patients exposed to high temperatures, especially when the humidity is high. The early symptoms include general malaise, yawning, headache and dizziness. The patient becomes restless, complains of severe pain in the head and chest, and consciousness is lost. The patient is flushed with a dry, hot skin. The temperature is elevated to 106° to 110° F. Early the pulse is full and rapid and the breathing deep and rapid. The pupils may be dilated early but later the pupils become constricted, the pulse is weak and irregular, and the respirations shallow and of the Cheyne-Stokes type.

Coma Due to Freezing.—Freezing as the cause of coma is indicated by the circumstances under which the patient is found. In this condition the patient's face is tranquil, the exposed parts are cold, stiff and pale, and pulse, respiration, temperature and blood pressure are low.

Hysterical "Coma."—Hysterical "coma" occurs in the presence of an audience. The patient assumes unusual or histrionic attitudes. The face is flushed, the pulse normal or rapid, the respirations slow, normal or rapid, and the blood pressure is normal. There is often fluttering of the eyelids, resistance to opening of the eyes, and rolling upward of the eyes when the latter is attempted. The reflexes are normal. Here again if the diagnosis is in doubt the history of a precipitating event may clarify the situation.

Fainting.—Simple fainting represents a stage of transient ischemia of the brain in response to a vasomotor depressor action. The patient is not in coma. The history of a precipitating cause, such as bad news, sight of blood, disagreeable odor, or other emotional stress aids greatly in making the diagnosis. The patient is pale, the pulse is rapid and feeble, the respirations increased, the blood pressure low and the pupils active. The short duration of the unconsciousness and the complete recovery makes the diagnosis evident.

ADDITIONAL ARTICLES

EXPERIENCES WITH STREPTOMYCIN

JAMES GOODFRIEND, D. PHIL. (OXON), M.D.*
AND DONALD L. THURSTON, M.D.*

WITH the isolation of streptomycin by Waksman and his associates,¹⁷ the spectrum of bacterial organisms against which antibiotic agents could effectively be employed was greatly widened. A vast literature has appeared detailing the properties of the new drug,^{2, 3, 10, 14, 19, 22} and numerous articles and reviews^{6, 19, 20, 41, 51, 59} have been published in an attempt to assess its clinical value. The consensus of the various investigators is that streptomycin has proved to be extremely effective in cases of infection with *Hemophilus influenzae*⁶ and with *B. tularensis*, that it is often valuable against infections with various susceptible organisms, particularly gram-negative bacillary infections of the genito-urinary^{2, 4, 12, 34, 35, 53} and respiratory tracts,²³ that it offers promise in some forms of tuberculosis,^{11, 14, 19, 20, 28, 42} and that it has been disappointing clinically in cases of infection with *Eberthella*^{22, 54} and *Salmonella*⁵⁶ organisms where in vitro results seemed to offer particular promise. The occasional toxic reaction to streptomycin as evidenced by impairment of vestibular function after prolonged administration and especially after intrathecal use or after very high blood levels have been maintained imposes a limiting consideration in its use which has not obtained in the case of penicillin.^{10, 15, 24, 32, 47} The usefulness of streptomycin is still further limited by the rapid acquisition of resistance by many different organisms.

DIARRHEA AND ENTERIC INFECTIONS

Diarrhea remains a major problem in pediatrics. In spite of improved methods of treatment, the disease still carries a high mortality. It is believed that the usual intestinal flora may become pathogenic in young or debilitated infants and that an ascending infection of the gastrointestinal tract with coliform organisms is a frequent finding. Since it has been shown⁵⁸ that streptomycin when given orally at least temporarily suppresses growth of the fecal flora, it might be supposed that oral streptomycin would be of value in treatment of the disease and that the upper gastrointestinal tract might be rid of coliform organisms by the drug. The fact that an appreciable number

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TABLE 1

PATIENTS WITH DIARRHEA AND POSITIVE GASTRIC CULTURES TREATED WITH ORAL STREPTOMYCIN

No.	Age	Duration Prior to Treatment (da.)	Duration Prior to Oral Strepto. (da.)	Treatment Other Than Strepto.	No. of Stools	Amt. Strepto. Orally (gm.)	Amt. Strepto. Subcu. (gm.)	Gastric Culture	Other Cultures	Duration of Diarrhea after Oral Strepto. (da.)	Blood Chemistry (Vols. %, mg./100 cc.)	Comments
1	3 da.	1	16	Pen., sulfon	8	1.455 16-30th da.	None	E. coli	Neg. bl. & stool	8	CO ₂ = 36.3 CO ₂ = 26.8 NPN = 55.0	Marked improvement after streptomycin. E. coli sensitive to 500 u./cc. streptomycin.
2	11 da.	7	7	None	Innumerable	0.7	0.9	E. coli	Stool, aerobac.; bl., neg.	3	CO ₂ = 23.0 Cl = 770.0	Epidemic diarrhea of newborn. Intracranial hemorrhage. Rapid response to treatment.
3	11 da.	2	6	Pen., sulfa	12	2.0 4-12th da.	None	E. coli	Stool, neg.; bl., neg.	2	CO ₂ = 18.0	Oral streptomycin gave blood level of < 5 and > 1 u./cc. No improvement until oral streptomycin started.
4	12 da.	3	5	Pen.	"Continuous"	1.5	0.22 on 5th dn.	E. coli	Bl., Staph. albus	5	CO ₂ = 16.6 pH = 6.98 Cl = 735.0	Infant feedings started on 4th day after treatment begun.
5	12 da.	6	13	Pen., sulfa	10-11	1.0 13-19th da.	0.95 6-13th da.	E. coli	Bl., neg.; stool, neg.	2	CO ₂ = 14.7 NPN = 57.0 pH = 7.1	Poor response to subcut. immediate response to oral streptomycin. Increased stools after strepto. stopped.
6	13 da.	7	9	None	8-15	1.0	1.0	E. coli	Stool, aerobac.; bl., neg.	4	CO ₂ = 28.2	Gastric culture neg. after 7 days.
7	13 da.	4	6	None	3	0.95	0.1	E. coli	Neg. bl. & stool	2	CO ₂ = 13.8	E. coli sensitive to 10 u./cc. strepto. Acute exacerbation on 9th hosp. day. Improves after 2 days of strepto.
8	3 wk.	6	12	None	4-6	0.36 12-15th da.	0.56 8-14th da.	E. coli	Neg. bl. & stool	1 (5 da. after subcu.)	CO ₂ = 40.5	No diarrhea in hosp. Epidemic diarrhea of newborn.
9	4 wk.	3	6	Pen., sulfa	3	0.56	0.34	E. coli	Bl., neg.	3	Not done	
10	6 wk.	30	40	Pen., sulfon	Frequent	0.92	0.6	E. coli	Neg. bl. & stool	5		
11	4 mo.	8	9	Pen., sulfa	5	1.38 1-10th da.	None	E. coli	Bl., neg.	No diarrhea in hosp.		

12	5 mo.	2	8	None	6-8	1.98 6-12th da.	1.44 5-11th da.	E. coli	Neg. bl. & stool	2 3d da. after Subcu.	CO ₂ = 39.5	Rapid improvement after oral streptomycin
13	6 mo.	11	11	Pen., sulfa	5-6	0.25	2.67 12-23d da. 1.3 30-34th da.	E. coli	Neg. bl. & stool	19	CO ₂ = 31.0	E. coli resistant to 10 u./cc. No response until anal drainage established.
14	6 mo.	14	16	Pen.	± 12	0.4	None	E. coli	Stool, B negative, gastric also contained staph. & aerobac.	2	Not done	No parenteral fluids required after streptomycin.
15	1 mo.	3	29	Pen	6-7	0.305	None	Staph.	Stool, neg.	4	CO ₂ = 40.8	Diarrhea first treated with penicillin. Oral strepto. given for relapse.
16	3 mo.	19	19	Sulfa, adrenal cortex	5-8	2.3	1.2	Staph	Not done	8	CO ₂ = 41.5	Acute catarrhal oilds modif. Ulcerative stomatitis.
17	6 wk.	Devel- oped on 13th hosp. da.	7	Sulfa, pen.	14	0.975 7-12th da.	0.1	E. coli on 8-11th- 13th da. of diar- rhea	Neg. bl. & stool	1	Gastric k. pH 4.5-5.11th da. 6.5 13th da.	Colon strains from gastric juice varied from < 3 u./cc. to > 10 u./cc. sensitivity Dfa made immediate response to strepto. orally although gastric cultures remained positive.

of cases of diarrhea are associated with bacteremia with organisms which are usually streptomycin-sensitive suggests that the drug may also be of value when administered parenterally.

A total of eighty-six cases of diarrhea have been treated with streptomycin administered orally or parenterally or by a combination of the two routes. These cases in no sense represent a controlled series. Because of the scarcity and cost of the drug, the younger patients and the more severely ill in general were the recipients of streptomycin. The results in this group of cases are summarized in Tables 1, 2, 3, 4 and 5. The cases have been grouped according to whether or not positive cultures were obtained from the gastric juice, according to the route of administration of streptomycin, and according to whether or not a bacteremia was demonstrated.

Table 1 contains summaries of seventeen cases in which microorganisms were cultured from the gastric juice and which received oral streptomycin with or without streptomycin administered subcutaneously. Of these, twelve were under 6 weeks of age. In these younger infants, *E. coli* was the organism obtained from the gastric juice in eleven cases, and a staphylococcus was recovered in the remaining case. The infants in general were severely ill and fell in the group which would ordinarily be given a very guarded prognosis. Eight of the patients were in severe acidosis, and two were in mild acidosis on admission. In two cases blood cultures were positive, in both instances the organism being different from that obtained from culture of the gastric juice. The number of stools varied from three per day to "innumerable" or "countless," and the stools were usually green and foul-smelling and contained mucus and occasionally blood. Eight patients received penicillin, and seven received sulfonamides in addition to streptomycin. Nine were treated with streptomycin by the subcutaneous as well as by the oral route. In addition all patients were treated with sodium lactate to correct the acidosis and with other parenteral fluids as indicated. All patients in this group recovered from the diarrhea. In six, decrease in number of stools and improvement in their character seemed to follow the administration of oral streptomycin in a dramatic fashion even though several of these had failed to respond to the drug when administered parenterally and to penicillin. In two, the diarrhea recurred when streptomycin was withdrawn. In one case the strain of *E. coli* isolated proved to be highly resistant, but the diarrhea subsided promptly. Sensitivity of the strains of *E. coli* isolated varied from 0.3 units of streptomycin/cc. to 500 units of streptomycin/cc. In the five older infants in the series, *E. coli* was recovered from the gastric juice in four cases, a staphylococcus in one. Acidosis was less marked and the diarrhea generally less severe than in the newborn group. In two cases in which there was parenteral infection, relief from the diarrhea was not obtained

TABLE 2

PATIENTS WITHOUT POSITIVE GASTRIC CULTURES TREATED WITH ORAL STREPTOMYCIN

No.	Age	Duration Prior to Treatment (da.)	Duration Prior to Oral Strepto. (da.)	Duration after Strepto. (da.)	Treatment Other Than Strepto.	No. of Stools	Amt. Strepto. Orally (gm.)	Amt. Strepto. Subcut. (gm.)	Gastro Culture	Other Cultures	Blood Chemistry (Vols. % mg./100 cc.)	Comments
18	2 wk.	7	8th hosp. da.	2	None	Indurated stool	0.545 8-13th da.	None	Not done	Stool, neg	Not done	Blood disappeared from stools, and stools decreased in number after streptomycin
19	3 wk.	4	4	8	None	Unknown	1.0	1.0	Not done	Stool, neg	CO ₂ = 15.0 pH = 7.0	Slow improvement with recurrence of diarrhea.
20	5 wk.	20	22	No diarrhea in hosp.	Pen., sulfa	5-14	0.475 2-4th da.	None	Not done	Stool, neg	Not done	Acute catarrhal otitis media.
21	6 wk.	4	4	6	Pen.	10-12	0.95 5-9th da.	1.7 4-8 da.	Not done	Bl neg	CO ₂ = 19.3 pH = 7.15	Good recovery with streptomycin.
22	2 mo.	28	30	8	Pen.	13-16	2.39 30-41st da.	2.79 50-41st da.	Not done	Stool, Aero-bac. alkali groes	CO ₂ = 39.5	Gradual decrease in fever and stools.
23	3 mo.	5	7	3	Pen.	6	1.4	None	Not done		CO ₂ = 49.0	Stools changed in 24 hr to normal character
24	5 mo.	30	32 44	See comment	Pen.	8	0.775 32-37th da. 0.1 44-48th da.	2.64 45-54th da.	Not done	Neg bl. & Stool	CO ₂ = 31.0 NPN = 45.0	Good initial response with relapse requiring 2nd course of streptomycin. Severe bronchitis.
25	6 mo.	14	14	4	Pen., sulfa	10-12	1.37 14-18th da.	1.33 14-17th da.	Not done	Stool, P. mirabilis, bl., neg.	CO ₂ = 43.0	Rapid recovery and gain in weight.
26	7 mo.	10	17	1	Pen., sulfa	8-10	0.55	None	Not done	Not done	Not done	Acute catarrhal otitis media developed on 6th hosp. day. Normal stools after 1st day of treatment.

TABLE 3

PATIENTS WITH DIARRHEA AND POSITIVE GASTRIC CULTURES NOT RECEIVING ORAL STREPTOMYCIN (SUBCUTANEOUS STREPTOMYCIN ONLY)

No.	Age	Duration of Diarrhea Prior to Adm. (da.)	Duration of Diarrhea Prior to Strepto. (da.)	Other Treatment	No. of Stools	Duration of Diarrhea after Strepto. (da.)	Amt. Strepto. Admin. (gm.)	Gastric Culture	Other Cultures	Other Lab.	Comments
27	4 da.	1	7	Pen., sulfa	5-9	11	2.12 7-21st da.	E. coli on 7th da.; neg. on adm.	Bl. cul., Staph. albus	CO ₂ = 34.6	Poor response to treatment with slow recovery.
28	7½ wk.	7	8	None	2	2	1.8	Staph. aureus	Neg. bl. cul.	CO ₂ = 21.0 pH = 7.3 NPN = 45.5 Cl = 629.0	Staph. sensitive to 0.5 u. streptomycin per cc.
29	2 mo.	6	8	Pen., sulfa	28	11	2.7	E. coli	Neg. bl. cul.	CO ₂ = 17.0	Marked response to streptomycin and relapse when drug was with- drawn.
30	15 mo.	3	5	Pen.	10-11	3	0.975	Strep.	Neg. bl. cul.	CO ₂ = 24.5 pH = 7.22 Cl = 700.0	Early response to treatment.

until the infection was eliminated. In two (12 and 14) rapid response to streptomycin was noted.

In Table 2 are summarized nine cases receiving oral streptomycin in which positive cultures of the gastric juice were not obtained. These infants varied in age from 2 weeks to 7 months with four falling in the neonatal age group. Six patients (18, 21, 23, 24, 25, 26) recovered rapidly from the diarrhea. In one (Case 24) a relapse was encountered after streptomycin was discontinued, but a favorable response to a second course of the drug was observed. In two cases, recovery was slow and not apparently associated with streptomycin administration, and in one no diarrhea was observed in the hospital.

Of four patients with positive cultures from the gastric juice (Table 3) who received subcutaneously administered but no oral streptomycin, two (27 and 29) showed a relatively delayed recovery. In both of these *E. coli* was grown from the gastric juice. In a case from which a staphylococcus and in another from which a streptococcus was cultured, recovery was rapid (Cases 28 and 30), although other therapy was less vigorous. In Case 29, diarrhea recurred after streptomycin was withdrawn.

Fourteen patients with diarrhea exhibited a concomitant bacteremia (Table 4). In eight of these the organism was a staphylococcus, in three a strain of *B. coli*. *Streptococcus viridans*, *B. proteus*, and a large gram-positive coccus were each recovered in one instance. The entire group was in the neonatal age range. Five were moderately to severely acidotic, and four were in mild acidosis. Two deaths occurred. In both fatal cases (Cases 35 and 43) streptomycin was begun on the day before death although the patients had been under treatment for five and three days respectively with penicillin. Death in these cases cannot fairly be described, therefore, as streptomycin failures.

Nine patients improved markedly within one to three days after streptomycin was begun. Of these, seven had apparently failed previously to respond to penicillin or penicillin and sulfonamides (Cases 33, 34, 36, 38, 41, 42, and 43). Two infants continued to pass diarrheal stools for five days after streptomycin was begun. One had no diarrhea after admission to the hospital. In one case diarrhea which had disappeared during streptomycin therapy recurred when the agent was withdrawn and again disappeared after streptomycin was restarted.

There were forty-four cases of diarrhea in which neither positive gastric juice nor blood cultures were obtained. Among these, one-half of the patients were under 6 weeks of age, and only one was over one year. In ten cases pathogens were isolated from the stools, umbilicus or nose and throat. The organism was most frequently a type of paracolon bacillus, but staphylococci, nonhemolytic streptococci, *Alcaligenes fecalis*, *pyocyaneus*, *E. coli*, and *proteus* were also encountered. Their significance is not entirely clear. At least three cases exhibited definite infection of the upper respiratory tract. In thirteen cases there was

TABLE 4

CASES OF DIARRHEA WITH POSITIVE BLOOD CULTURE RECEIVING STREPTOMYCIN (SUBCUTANEOUSLY)

No.	Age	Duration of Diarrhea Prior to Adm. (da.)	Duration of Diarrhea Prior to Strepto. (da.)	Other Treatment	No. of Stools	Duration of Diarrhea after Strepto. (da.)	Amount Strepto. Adm. (gm.)	Blood Culture	Other Lab. (mg./100 cc.)	Comments
31	4 da.	2	18	Pen.	8-10	No diarrhea in hosp.	1.7 18-22nd da.	Strept. viridans	Not done	Epidemic diarrhea of newborn. Sepsis without symptoms.
32	6 da.	1	1	Pen.	12	5	0.52 1-5th da.	Staph. aureus	CO ₂ = 29.0	Apparent rapid response.
33	2 wk.	2	25	Pen., sulfa, adrenal ext.	9	3	1.885 50,000 I. T.	Staph. albus	E. coli from CSF CO ₂ = 20.0	Strepto. given for colon meningitis with almost immediate cessation of severe diarrhea
34	10 da.	2nd hosp. da. (cross infection)	8	Pen., sulfa	11-12	No diarrhea during strepto. admn.	3.75 7-20th da.	Staph. albus	Not done	Staph. sensitive 0.05-0.1 units of strepto. and 5 u./cc. penicillin
35	2 wk.	5	10	Pen.	8	Died on 11th day of disease	0.17	E. coli	CO ₂ = 22.0 NPN = 120.0	Anuria. Terminal pneumococcus sepsis.
36	17 da.	2	24	Pen., sulfa	9	1	0.2	E. coli	CO ₂ = 27.0	Improved on day streptomycin started.
37	6 wk.	1	6	Pen., sulfa	6	5	1.73	Staph. aureus	Not done	Staph. sensitive to 0.5 u./cc. strepto. Neg. stool culture.
38	3 mo.	5	10	Pen., sulfa	10-12	2	1.41 10-16th da.	Large gram-pos. coccus	CO ₂ = 35.7	Possible contaminant in blood culture. Improved with streptomycin.
39	9 da.	1	1	Pen.	2	1	0.96	Staph. albus	CO ₂ = 40.0 Nose and throat, Staph. albus	Staph. from nose resistant to strepto. 15 u./cc. to penicillin 0.4 u./cc.
40	5 da.	1	1	Pen.	2	2	1.92	Hem. Staph. albus	CO ₂ = 10.0 Throat cul. diphtheroid	Relapse of diarrhea after cessation of streptomycin with response to readministration.
41	11 da.	1	4	Pen., sulfa	7	1	1.6	Staph. albus	CO ₂ not done E. coli from throat	Failure of response to penicillin and sulfa. Good response to streptomycin.
42	6 da.	1	5	Pen., sulfa	2	3	1.6	E. coli	CO ₂ = 38.0 NPN = 24.0 Cl = 441.0	Throat culture contained pneumococcus.
43	5 da.	1	5	Pen., sulfa	2	1½	1.6	H. proteus resist pen., sensitive to strepto.	CO ₂ = 36.0	Penicillin and sulfa no effect. Rapid response to strepto.
44	4 da.	1	4	Pen., sulfa	1	Died 5th hosp. day		Staph. albus, H. & non-H. indiff. strepto.		

mild acidosis, and in thirteen the acidosis was moderate to severe. In eight cases, recovery from diarrhea as measured by decreased number of stools, improvement in their quality, and increased ease of maintaining hydration followed within one to three days the beginning of streptomycin treatment after treatment with penicillin and sulfonamides had been unsuccessful over periods varying from two to ten days. In another case recovery required five days. The diarrhea recurred in three patients after streptomycin was discontinued and began in one on the day streptomycin was discontinued. In eight cases no diarrhea was noted during the hospital stay, although diarrhea was the patient's presenting complaint. In eighteen cases in which both penicillin and streptomycin were begun simultaneously rapid recovery from diarrhea was observed. In five cases response must be considered poor. In one of these death occurred after the patient had received penicillin and sulfonamides for fifteen days and streptomycin for five days. The course was protracted in another case in which streptomycin was given for only two days (Case 9). In a third, diarrhea recurred intermittently until an infected temporal bone was treated surgically. In one case, diarrhea developed while the patient was receiving streptomycin for another illness.

Although no control series is available and no exact comparisons with previous series is valid because of the great variability in severity of diarrhea from year to year and the dissimilarity between cases, the conclusion cannot be escaped that the results in this series have been excellent both from the point of view of low mortality and of speed of recovery. Only three deaths (3.5 per cent) occurred in the series, and of these, two occurred in cases in which streptomycin had been utilized only on the last day of life. The number of cases in which cessation of diarrhea occurred immediately after institution of streptomycin treatment and the number of cases in which recurrence of diarrhea followed discontinuance of the drug is probably too large to be considered entirely coincidental. Although more study is required to establish the point, it appears that streptomycin constitutes an important adjunct to the treatment of infantile diarrhea and that oral streptomycin is probably of value in cases in which colon bacilli have invaded the stomach. The medium in which oral streptomycin should be administered and the time it should be given in relation to meals must be determined in the light of knowledge that its effectiveness is altered by changes in pH and decreased in the presence of dextrose.²¹

Although experimental studies with salmonella organisms^{21, 22} have generally indicated good sensitivity to streptomycin, clinical response to the drug both in the active disease and in the carrier state has been disappointing.²³ Some investigators, however, have obtained fair response in these cases.²² Of nine cases (Table 6) of *salmonella infection*, fair to good results were obtained in six with streptomycin treatment. In one case (Case 93) clinical improvement was noted with

TABLE 4

CASES OF DIARRHEA WITH POSITIVE BLOOD CULTURE RECEIVING STREPTOMYCIN (SUBCUTANEOUSLY)

No.	Age	Duration of Diarrhea Prior to Adm. (da.)	Duration of Diarrhea Prior to Strepto. (da.)	Other Treatment	No. of Stools	Duration of Diarrhea after Strepto. (da.)	Amount Strepto. Admin. (gm.)	Blood Culture	Other Lab. (Vols. % mg./100 cc.)	Comments
31	4 da.	2	18	Pen.	8-10	No diarrhea in hosp.	1.7 18-22nd da.	Strept. viridans	Not done	Epidemic diarrhea of newborn. Sepsis without symptoms.
32	6 da.	1	1	Pen.	12	5	0.52 1-5th da.	Staph. aureus	CO ₂ = 29.0	Apparent rapid response.
33	2 wk.	2	25	Pen., sulfa, adrenal ext.	9	3	1.885 50,000 I. T.	Staph. albus	E. coli from CSF CO ₂ = 20.0	Strepto. given for colon meningitis with almost immediate cessation of severe diarrhea
34	10 da.	2nd hosp. da. (cross infection)	8	Pen., sulfa	11-12	No diarrhea during strept. to admn.	3.75 7-20th da.	Staph. albus	Not done	Staph. sensitive 0.05-0.1 units of strepto. and 5 u./cc. penicillin
35	2 wk.	5	10	Pen.	8	Died on 11th day of disease	0.17	E. coli	CO ₂ = 22.0 NPN = 120.0	Anuria. Terminal pneumococcus sepsis.
36	17 da.	2	24	Pen., sulfa	9	1	0.2	E. coli	CO ₂ = 27.0	Improved on day streptomycin started.
37	6 wk.	1	6	Pen., sulfa	6	5	1.73	Staph. aureus	Not done	Staph. sensitive to 0.5 u./cc. strepto. Neg. stool culture.
38	3 mo.	5	10	Pen., sulfa	10-12	2	1.41 10-16th da.	Large gram-pos. coccus	CO ₂ = 35.7	Possible contaminant in blood culture. Improved with streptomycin.
39	9 da.	1	1	Pen.	2	1	0.96	Staph. albus	CO ₂ = 40.0 Nose and throat, Staph. albus	Staph. from nose resistant to strepto. 15 u./cc. to penicillin 0.4 u./cc.
40	5 da.	1	1	Pen.	2	2	1.92	Hem. Staph. albus	CO ₂ = 10.0 Throat cul. diptheroid	Relapse of diarrhea after cessation of streptomycin with response to readministration.
41	11 da.	1	4	Pen., sulfa	7	1	1.6	Staph. albus	CO ₂ not done E. coli from throat	Failure of response to penicillin and sulfa. Good response to streptomycin.
42	6 da.	1	5	Pen., sulfa	2	3	1.6	E. coli	CO ₂ = 38.0 NPN = 24.0 Cl = 441.0	Throat culture contained pneumococcus.
43	5 da.	1	5	Pen., sulfa	2	1½	1.6	B. proteus resist pen., sensitive to strepto.	CO ₂ = 36.0	Penicillin and sulfa no effect. Rapid response to strepto.
44	4 da.	1	4	Pen., sulfa	1	Died 5th hosp. day		Staph. albus, H. & non-H. indiff. strepto.		

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TABLE 5

DIARRHEA PATIENTS WITHOUT POSITIVE GASTRIC OR BLOOD CULTURES TREATED WITH STREPTOMYCIN (SUBCUTANEOUSLY)

No.	Age	Duration Prior to Treatment (da.)	Duration Prior to Streptomycin (da.)	No. of Stools	Other Treatment	Amount of Streptomycin (gm.)	Lab. (Vols. % mg./100 cc.)	Duration of Diarrhea after Streptomycin (da.)	Comments
45	14 hr.	5	15	1	Pen., sulfa	1.25 15-25th da.	CO ₂ = 53.0 Prot. = 3.63	—	Died 20th day. Autopsy showed necrotizing colitis and central hepatic necrosis.
46	3 da.	1	3	3	Pen., sulfa	2.0	Not done	1	Paraurthral redness. Good response to streptomycin.
47	4 da.	1	1	1	Pen.	0.3	Not done	1	Rapid response.
48	4 da.	1	9	3	Pen., sulfa	2.1	CO ₂ = 37.0	2	Had two respiratory infections without recurrence of diarrhea.
49	5 da.	1	9	2	Pen., sulfa	2.0	CO ₂ = 28.0	2	Stools contained a penicillin-resistant paracolobacillus.
50	6 da.	1	2	5	Pen.	0.3	CO ₂ = 38.0 NPN = 24.0 Cl = 441.0	1	Improved within 24 hr.
51	7 da.	1	1	5	Pen., sulfa	2.0	NPN = 21.0 Cl = 479.0 CO ₂ = 37.0	1	Throat and stool contained nonhemolytic strep. and paracolob. Pen. and sulfa discontinued on 5th day. Streptomycin maintained.
52	8 da.	1	11	1	Pen., sulfa	1.6	CO ₂ = 34.0	2	Throat contained E. coli. Rapid improvement and ability to take food 2 days after streptomycin.
53	9 da.	4	5	5-7	Pen., sulfa	0.82 5-10th da.	CO ₂ = 22.5	2	Bl. neg. Stool contained paracolob. Rapid recovery without relapse.
54	10 da.	3	5	1-5	Pen.	0.66 4-9th da.	CO ₂ = 36.5	3	Rapid response.
55	2 wk.	5	5	Numerous	Pen.	0.84 5-11th da.	CO ₂ = 29.5 Cl = 790.0 NPN = 71.0	3	Acidosis and shock. Good response.
56	2 wk.	5	5	25-30	Pen.	0.465 5-10th da.	CO ₂ = 53.0	4	Good response.
57	2 wk.	1	4	12-14	Pen.	0.9	CO ₂ = 38.0	5	E. coli from penis and umbilicus. Jaundice and dehydration.
58	17 da.	8	10	6	None	1.42 2-7th da.	Not done	See comment	No diarrhea in hospital.
59	3 wk.	2	2	20-25	Pen.	0.225 1st & 2nd	CO ₂ = 41.0	7	Streptomycin given only 2 days.

60	3 wk.	2	2	Frequent	Pen.	0.44 1-14th da.	CO ₂ = 37.5	See comment	No diarrhea for first 3 hosp. days. Diarrhea with blood began on day streptomycin was discontinued.
61	1 mo	10th hosp. da.	21	12	Pen., adrenal	0.42	Not done	2	Ptyococcus from stool sensitive to > 0.1 u./cc. penicillin and > 0.5 u./cc. strepto
62	1 mo.	4	6	7	Pen.	0.38 6-11th da.	Not done	3	Blood and stool neg. Rapid recovery from bloody diarrhea.
63	1 mo.	20	20	Innumerable	Pen.	2.4 20-40th da.	CO ₂ = 49.6	3	lit. neg. Stool contained Alcaligenes (ecoli. No fluid required after 5th day of treatment.
64	4 wk.	21	22	7	Pen., sulfa	0.775 22-25th da.	CO ₂ = 43.8	3	Cultures not done. Good recovery on treatment.
65	1 mo.	7	7	3-4	Pen.	0.46 7-14th da.	CO ₂ = 53.0	3	Cultures not done. Prompt recovery and defervescence after treatment started.
66	6 wk	Started on 6th hosp. da.	8 da. before diarrhea	11-12	Pen., sulfa	2.16 1-16th da.	Not done	See comment	Developed diarrhea on streptomycin
67	2 mo.	9	10	10-20	Noose	1.44	CO ₂ = 35.3 pH = 7.15	8	Y-chills 2 days. Maintained hydration after 3d day
68	2 mo.	13	13	3	Sulfa	2.0	CO ₂ = 20.0 NaCl = 752.0	3	Myeloma coagula.
69	2 mo.	8	8	12-15	Sulfa	1.17 8-14th da.	CO ₂ = 32.6	2	Rapid clinical improvement noted.
70	2 mo.	3	3	8	Noose	0.98 1-7th da.	CO ₂ = 18.0 Cl = 748.0	4	Rapid improvement with streptomycin.
71	2 mo.	8	9	3	Pen., sulfa	0.775 9-15th da.	CO ₂ = 48.8	3	No fluids required after 3 days.
72	2 mo.	4	5	7-8	Pen., sulfa	1.36 8-11th da.	CO ₂ = 17.8 pH = 7.2 NPN = 91.0	5	No fluids required after 5th day Temp. fell to normal within 36 hr of beginning of therapy
73	2 mo.	5	6	4	Pen.	2.25 6-17th da.	CO ₂ = 25.7	See comment	No diarrhea in hospital. P. marginal obtained from stools.
74	10 wk.	4	6	Few	Pen., sulfa	0.7 2-7th da.	CO ₂ = 33.1 Cl = 591.0	See comment	No diarrhea in hospital
75	4 mo.	14	15	4-5 bloody	Noose	0.34 1-3d da.	CO ₂ = 26.7	See comment	No diarrhea in hospital

streptomycin after the drug was administered orally although the stools remained infected. Results must be classified as poor in three cases. In one of these (Case 90) the stools remained infected after a course of streptomycin administered subcutaneously. In another (Case 95) streptomycin proved ineffective, and clinical recovery coincided with the administration of sulfonamides. One case died unexpectedly while undergoing treatment. It is probable that the death was not attributable to streptomycin toxicity or to the paratyphoid infection. Although these results are equivocal and *in vitro* tests of sensitivity probably are not suitable gauges of expected clinical response, the occasional favorable result of streptomycin therapy justifies its clinical trial in this disease.

MENINGITIS

Experiences with streptomycin in the treatment of *influenzal meningitis* have not differed from those reported elsewhere. Alexander,^{7, 9} Birmingham and associates¹² and others^{44, 52, 63} have emphasized that early in the course of the disease streptomycin alone given subcutaneously and intrathecally is usually adequate for cure. In young infants, in long-standing cases, and in infections with resistant organisms or with organisms which have become resistant, type-specific antiserum and sulfonamides must be added to the therapeutic regimen.

Streptomycin has not been used alone in the treatment of meningitis due to *H. influenzae* at the St. Louis Children's Hospital, but all agents of possible therapeutic value have been employed in each case. Because of the frequency of cases in which the identification of the organism is impossible or uncertain on examination of the initial spinal fluid specimen, because of the impossibility of rapid determination of bacterial sensitivity to the agent and because of the occasional case of meningitis due to mixed infection with *H. influenzae* and other organisms, it has been our practice to administer penicillin and streptomycin intrathecally at the time of the initial lumbar puncture when cloudy cerebrospinal fluid is first obtained. Treatment by subcutaneous administration of both antibiotic agents and occasionally with sulfonamides is begun simultaneously. Every effort is made to identify the infecting organism and determine its sensitivity as rapidly as possible. The spinal fluid is inoculated not only on the usual media but on the developing chick embryo as well. Cultures of the blood are obtained before any treatment is begun. The inclusive treatment is continued until the organism is identified and its sensitivity to penicillin, streptomycin and sulfonamides determined. The ineffective therapeutic agents are then discontinued.

Results have appeared to justify this initial broad antibacterial coverage. Since streptomycin has become generally available, twenty-five cases of acute meningitis have been handled in this manner. Of

these, ten proved to be meningitis due to *H. influenzae*. Brief summaries of these cases follow.

CASE 1—S. D., a 10 months old white female, was admitted after a one month's illness which had been diagnosed elsewhere as pneumococcus meningitis and treated for one week with sulfamerazine. Fever had continued, but pleocytosis decreased. The patient had received 20,000 units of penicillin intrathecally daily for three days. Physical examination revealed an ill child with full fontanelle. A lumbar puncture was performed and 20,000 units of penicillin and 30,000 units of streptomycin were instilled into the subarachnoid space. The infant was treated with streptomycin, penicillin and sulfonamide drugs from the day of admission. After twenty-four hours an organism identified as *H. influenzae* was obtained from the cerebrospinal fluid culture and penicillin was discontinued. Since on the third day no quelling of the organism was noted with the patient's serum 100 mg of specific antiserum (*H. influenzae* type B) were administered, and on the following day quelling was obtained. The patient received 0.9 gm streptomycin intrathecally from the first to twelfth hospital day and 11.15 gm subcutaneously from the first to the nineteenth day. The temperature remained normal after the fourth hospital day. The number of cells in the cerebrospinal fluid gradually decreased from 500 to 12/cu mm by the twenty-fourth day. No growth was obtained on culture after the initial specimen, and the cerebrospinal fluid sugar rose from 37.2 to 136 mg. per 100 cc on the sixth day. By this time the Pandy had become negative. A pneumoencephalogram on the thirty-second hospital day revealed slight ventricular dilatation. The patient appeared alert and in good condition. This represents an old neglected case with delayed treatment in a young infant. The response was dramatic and recovery apparently complete.

CASE 2—W. K., a 0 weeks old infant, had vomited since birth. His head had grown more rapidly than normal. On examination the head measured 40 cm., and the fontanelle bulged. A small amount of yellow turbid cerebrospinal fluid was obtained from the ventricles; the glucose content was 10 mg per 100 cc., and *H. influenzae* was identified on smear. Twenty thousand units of penicillin and 50,000 units of streptomycin were instilled into the ventricles. Sulfadiazine and serum were administered. Intrathecal streptomycin (50,000 units) was given for four days, and a total of 3.36 gm of streptomycin was given subcutaneously. The spinal fluid cell count fell from 640 to 20 on the ninetieth day. Pneumoencephalogram showed marked ventricular dilatation, and the infant was obviously spastic and hydrocephalic. He expired sixty-two days after his original admission following an exploratory craniotomy.

CASE 3—W. T., a 15 months old infant, had been ill for twelve hours prior to admission. A lumbar puncture was performed and streptomycin 50,000 units and penicillin 20,000 units were instilled into the lumbar subarachnoid space. *Hemophilus influenzae* was identified on smear of the cerebrospinal fluid. Seventy-five milligrams of specific antiserum were given and the patient placed on streptomycin, penicillin and sulfadiazine. A total of 0.70 gm of streptomycin was administered intrathecally during the first eight days, and 7.625 gm. were given subcutaneously during a like period. The cell count of the cerebrospinal fluid fell from an initial level of 5300 to 8 on the eighth day. The original spinal fluid glucose level was 18 mg per 100 cc. The organism was cultured from the cerebrospinal fluid on each of the first three days. Temperature dropped critically to normal levels after fifty-two hours, and the patient was discharged on the fifteenth day without sequelae.

CASE 4—S. H., 10 months old, was admitted with a twenty-day history of fever and opisthotonus, and convulsions for one week. Treatment with sulfathiazole had

been instituted. Kernig and Brudzinski signs were positive; deep reflexes were hyperactive, and the fontanelle bulged. On admission 20,000 units penicillin were instilled intrathecally, and eight hours later 20,000 units of penicillin and 50,000 units of streptomycin were administered by the same route. An organism which was quelled by *H. influenzae* antiserum, type B, was identified. The patient was started on sulfonamides, penicillin and streptomycin subcutaneously, and 75 mg. of type B antiserum were administered. Streptomycin 0.45 gm. was given intrathecally during the first five days and 4.9 gm. subcutaneously during the same period. Except for irregular athetoid movements one to two hours after intrathecal streptomycin on the third and fourth days, recovery was uneventful. The patient was afebrile after the sixth day. The cerebrospinal fluid cell count fell from 74,800 to 45; the Pandy became negative; *H. influenzae* was not seen or cultured after the third day, and the cerebrospinal fluid glucose rose from 5 mg. per 100 cc. on admission to 47 mg. per 100 cc. There were no evident sequelae.

CASE 5.—A. R. was a 3 year old white female who had had stiff neck and stupor for one day. Sulfonamides had been administered. On physical examination bilateral otitis media and meningeal irritation were noted. When cloudy fluid was obtained on lumbar puncture, antibiotics were instilled into the subarachnoid space and administered subcutaneously. In addition, sulfonamides and antiserum were given, although the quellung test was positive on admission. The patient appeared entirely normal after the third day. The cell count in the cerebrospinal fluid fell from 7500 to 201 by the sixth day; Pandy became negative; *H. influenzae* was not found or cultured from the cerebrospinal fluid after the second day, and the patient recovered without incident. Streptomycin 0.250 gm. was given intrathecally from the first to the sixth day, and a total of 4.72 gm. had been given subcutaneously.

CASE 6.—L. K., a 3 years 2 months old white male, was admitted on the third day of an illness manifested by vomiting, fever and convulsions. He had received 100,000 units of penicillin intrathecally. There were signs of meningeal irritation. *Hemophilus influenzae* was identified on our original smear of the cerebrospinal fluid and streptomycin given intrathecally and subcutaneously. Type B antiserum was also administered (75 mg). In all, 4 gm. of streptomycin were given subcutaneously and 0.050 gm. intrathecally during the first ten days. After the first day the cerebrospinal fluid was sterile. The cell count fell from 2640 to 29, and the patient made a rapid recovery within four days of admission. On discharge there was no evidence of sequelae.

CASE 7.—C. K., 3 years 5 months of age, was admitted on the fifth day of illness. He had received penicillin intrathecally and subcutaneously and sulfadiazine at another hospital where *H. influenzae* was reported in the cerebrospinal fluid. No organism was recovered at this hospital. A single dose of 50,000 units of streptomycin was given intrathecally, and 1 gm. of streptomycin subcutaneously was administered during the first three days. Sulfadiazine and penicillin were also employed. Recovery was rapid and uneventful. The patient was discharged after sixteen days in good condition. The cerebrospinal fluid cell count had fallen from 722 to 52.

CASE 8.—J. S. was 3 years 10 months old on admission. His illness had begun ten days before admission and had progressed, so that he was in marked opisthotonus. Treatment with penicillin and streptomycin intrathecally was begun immediately, and penicillin and streptomycin as well as sulfonamides were administered subcutaneously. *H. influenzae* was not recovered until the third hospital day by which time treatment had already effected an almost complete cure. No serum was used. A total of 0.3 gm. of streptomycin was given intrathecally during

the first six days, and 3.6 gm. were administered subcutaneously during the first nine days. The spinal fluid was normal by the sixth day, and the patient made a complete recovery.

CASE 9.—B. R. was a 7½ months old white female who had had fever and signs of meningeal irritation including convulsions for two weeks before admission. She had received sulfadiazine and a single daily injection of penicillin for twelve days. Physical examination revealed a very ill infant with bilateral suppurative otitis media and full fontanelle. Twenty thousand units of penicillin and 40,000 units of streptomycin were given intrathecally on the initial lumbar puncture, and subcutaneous administration of both antibiotics and sulfonamides were begun. A gram negative pleomorphic rod was identified on smear of the cerebrospinal fluid obtained on the first lumbar puncture, but the following day a pneumococcus type 19 was grown from the blood, and an identical pneumococcus was recovered on culture of the cerebrospinal fluid in addition to *H. influenzae*. All therapy was continued in view of the mixed infection. A total of 0.84 gm. of streptomycin was administered intrathecally from the first to twelfth day during which period penicillin was also given. The temperature remained normal after the sixth day, and the cerebrospinal fluid was sterile after twenty four hours of treatment.

CASE 10.—F. H., age 14 months, was admitted with a three weeks' history of fever, drowsiness and vomiting. An organism identified as *H. influenzae* was found on smear of the cerebrospinal fluid but could not be cultured. Streptomycin 50,000 units and penicillin 20,000 units were given intrathecally for the first four days, and both antibiotic agents were continued subcutaneously for seven days. Recovery was uneventful and complete.

Cases 5, 6, and 7 which occurred in older children who were treated relatively early in the course of the disease might well have been expected to recover with any good therapeutic regimen. The other cases, however, impose a sterner test of the adequacy of the treatment plan.

Cases 1, 4, 8, 9 and 10 illustrate the efficacy of streptomycin in this disease even in the face of prolonged delay (ten to thirty days) in treatment or inadequate treatment. This is especially impressive in Case 1, a 10 months old infant virtually untreated for a month. Case 2 represents the only fatality in the series. In this instance recovery from meningitis had occurred, but the complications of meningitis (hydrocephalus, cortical atrophy) which had been present before streptomycin was employed remained.

In Cases 1 and 8 there were lapses of twenty-four and seventy-two hours respectively between the admission treatment and identification of the organism. The early institution of therapy can only have been of benefit to the patient in view of the known worsening of prognosis as to recovery and sequelae with delay in treatment.

The immediate administration intrathecally of both penicillin and streptomycin when cloudy cerebrospinal fluid is obtained is best justified by the findings in Case 8 where a mixed *H. influenzae*-pneumococcus infection was found. Use of both antibiotic agents intrathecally provided adequate coverage against both organisms from the initiation of treatment although more than a day elapsed before the pneumococcus was isolated and identified.

Of the remaining cases of acute meningitis five either were caused by organisms which were shown to be streptomycin sensitive or responded clinically to streptomycin. One case of *meningitis due to E. coli* (Case 11) was encountered.^{5, 17, 60} This occurred during the course of severe diarrhea associated with *E. coli* sepsis. On the twenty-second day of illness this month old infant who had previously been treated intensively with penicillin and sulfa was found to have cloudy spinal fluid with 5200 cells. *Escherichia coli* was cultured from the fluid. On the first day of meningitis 50,000 units of streptomycin were placed in the lumbar subarachnoid space, and the drug was administered subcutaneously. In all, the baby received four doses of streptomycin intrathecally during the first five days and 2 gm. of streptomycin subcutaneously over a ten day period. The cell count in the cerebrospinal fluid fell rapidly; cultures were sterile after the first day, and the strain of *E. coli* was found to be sensitive to less than 0.5 unit/cc. of streptomycin. Temperature became normal, and diarrhea markedly improved after streptomycin was begun.

Two cases of *staphylococcic meningitis* responded to streptomycin therapy. The first (Case 12) was in a 14 year old boy who had sustained a skull fracture and developed meningitis which was treated for twenty-four days with penicillin and sulfadiazine. The course remained unfavorable until the twenty-fifth day of the disease when intrathecally and subcutaneously administered streptomycin was begun. The patient received 0.2 gm. of streptomycin intrathecally during a four day period and 10.8 gm. subcutaneously. Coincident with the beginning of streptomycin therapy the temperature subsided and remained normal. The child was discharged in a markedly stuporous state.

Case 13 was that of a 10 day old infant who had developed fever eight hours before admission. On the first hospital day opalescent cerebrospinal fluid containing 900 cells per cubic mm. was obtained by lumbar puncture. Streptomycin (50,000 units) and penicillin (20,000 units) had been administered intrathecally at the time of the lumbar puncture. A staphylococcus was cultured from the blood and nasopharynx which proved to be sensitive to 0.1 to 0.5 unit/cc. of penicillin and to 0.5 unit/cc. of streptomycin. The patient received in all 4.4 gm. of streptomycin and 2,350,000 units of penicillin and completely recovered although a urinary infection with a streptomycin-resistant *E. coli* developed during the course of streptomycin treatment.

Case 14 was due to a gamma streptococcus sensitive to 0.5 unit/cc. streptomycin. The drug was not employed in this case. Case 15 was an acute meningitis which followed injury to a pilonidal cyst. No organism was isolated, but treatment with streptomycin, penicillin and sulfonamides was employed in the usual dosage, and rapid recovery ensued.

It is unfortunate that streptomycin was not used initially in Case 12.

Earlier control of the meningeal infection might well have altered the outcome in this child. This case and Case 13 illustrate the fact that organisms which are usually streptomycin resistant may in some strains be sensitive. The determination of streptomycin sensitivity before streptomycin is discontinued is, therefore, emphasized. The desirability of initial employment of streptomycin is demonstrated by Case 14 in which infection with a sensitive organism was not treated with streptomycin.

Six cases of meningitis treated initially with streptomycin, penicillin and sulfonamides proved to be due to *pneumococcal infection*. The etiologic agent was established by culture within forty-eight hours of admission in all of these, and streptomycin was discontinued. Three cases of meningococcus meningitis were similarly handled as was a case of meningitis due to a gamma streptococcus which was found to be streptomycin resistant.

In none of the patients who suffered from meningitis due to resistant organisms was any toxic reaction or other ill effect noted with the brief initial course of streptomycin.

SEPTICEMIA

Twelve cases of septicemia or bacteremia which were treated with streptomycin are summarized in Table 7. Of these, nine were infections with staphylococci. These varied in severity from cases of bacteremia without clinical manifestation to severe sepsis with multiple localizations in bone, joints, brain and pericardium. Six of the strains of staphylococcus were very sensitive to streptomycin; two were insensitive, and one was not tested. Three of the strains were relatively resistant to penicillin, but all of these were streptomycin sensitive. In four of the cases, initial treatment consisted of penicillin and sulfonamides without improvement in the patient. In all of these, rapid recovery followed institution of streptomycin therapy. In two others recovery should probably be attributed to penicillin. In the case in which streptomycin sensitivity was not tested, the clinical course was such as to suggest strongly that streptomycin was an important factor in producing recovery.

Two cases of blood stream invasion with *E. coli* were encountered. In one (Case 10) there was an associated pneumococcus infection; streptomycin was not begun until the third day, and the course was rapidly downhill to a fatal termination. In the other, many episodes of transient *E. coli* bacteremia in a patient in the nephrotic stage of glomerulonephritis seemed to respond more rapidly and completely to streptomycin than to sulfonamide or penicillin treatment. In the last case, *Strep. fecalis* was the infecting organism, and blood stream invasion occurred while the patient was receiving large amounts of penicillin.

It is felt that these data suggest the advisability in cases of sepsis

TABLE 7

CASES OF SEPTICEMIA TREATED WITH STREPTOMYCIN

No.	Age	Organism Obtained from Bl. Culture	Streptomycin Sensitivity	Penicillin Sensitivity	Treatment (total)	Duration after Streptomycin	Localization of Infection and Clinical Manifestations	Comments
1	1 mo.	Staph. alb.	0.5 u./cc.—growth 2.0 u./cc.—no growth	1.0 u./cc.—growth 5.0 u./cc.—no growth	2.1 gm. strepto., 2.1 gm. pen.	Bl. culture neg. on 4th da.; temp. normal after 5th da.	Bacteremia without clinical manifesta- tion	Failure of response to sulfa and pen. and fairly prompt response to strepto.
2	1 mo.	Staph. alb.	Sensitive to 0.5 u./cc.		2.55 gm. strepto. 1-8th da.	Bl. cultures pos. for 1st 5 da.	Bacteremia without clinical manifesta- tion	Gradual disappearance of staphylococci from blood stream.
3	9 da.	Staph. alb.	Sensitive to 0.5 u./cc.	Sensitive to 0.5 u./cc.	4.5 gm. strepto. 2-12th da.	Bl. culture neg. on 4th da. and thereafter	Meningitis, pyelitis	Complete recovery.
4	4 mo.	Staph. alb.	"Very sensitive"	Resistant to 5.0 u./cc.	1 gm. strepto. 44th da.	4 da.	Fever and diarrhea	Persistent bacteremia and fever not re- sponding to sulfa or pen. but responding immed. to 1 day's treatment with strepto.
5	19 da.	Staph. alb.	Insensitive	0.1 u./cc.	0.6 gm. strepto. 1-5th da., 520,000 pen. 1-6th da.	5 da.	Fever and restlessness	Streptomycin ineffective. Penicillin prob- ably responsible for cure.
6	13 da.	Staph. alb.	Insensitive	0.1 u./cc.	1.2 gm. strepto. 2-5th da., 600,000 u. pen.	6 da.	None	Erythroblastosis fetalis. Recovery should be attributed to penicillin.
7	18 da.	Staph. aureus	Not done	Not done	3.2 gm. strepto. 4-8th da., 4.8 gm. 11-17th da., pen. throughout	Fall in temp. with 1st course of strepto., relapse after drug re- moved, rapid re- sponse to 2nd course	Arthritis rt. elbow & knee, osteomyelitis rt. femur and hum- erus, pyelonephritis, brain abscess, RLL pneumonia	Treated initially with sulfonamides and pen. without marked benefit. Immediate response to two courses of strepto. with relapse on discontinuance of 1st course.

8	1 mo.	Staph. aureus	0.5 u./cc.	Resistant to 0.5 u./cc. Sensitive to 1.0 u./cc. Resistant to 1.0 u./cc. Sensitive to 5.0 u./cc.	3.05 gm strepto. 22-30th da.	3 da.	Fever and diarrhea	Infant failed to respond to pen. and sulfoamides. Deference and recovery followed by 3 days institution of strepto. therapy
9	6 yr	Staph. aureus	0.5 u./cc. (strepto level 1 hr after admin. = 10 u./cc)	Resistant to 0.1 u./cc. Sensitive to 0.5 u./cc.	22.55 gm. strepto. 1-21st da., pen. (for same period)	No cultures pos. after 1st da.	Osteomyelitis rt. tibia, acute periocarditis, septic arthritis (rt. knee)	Rapid response of very severe septicemia. Sterilization of purulent exudate in rt. knee and rt. tibia.
10	17 da.	E. coli pneumococcus	Not done	Not done	0.17 subcu. 0.005 I.V. 3 & 4th da., pen	Died on 4th da.	Jaundice, diarrhea	Overwhelming sepsis. Pt. not treated immediately with strepto.
11	4 yr	E. coli	0.5 u./cc.		Repeated courses (7) over 5 mo. period	5-7 da.	Chronic glomerulonephritis, developing frequent episodes of E. coli bacteremia & meningitis	Usually prompt response.
12	7 mo.	Strep. fecalis	Resistant to 0.5 u./cc. Sensitive to 2.0 u./cc.	Resistant to 5.0 u./cc.	5.55 gm. strepto. 3-8th da.	2 da.	Fever and diarrhea	Symptoms developed while pt. was receiving massive doses of penicillin for congenital syphilis. Rapid recovery after streptomycin begun.

of initiating treatment with the broadest possible antibacterial coverage and continuing such treatment until the responsible organism is isolated and its sensitivities determined. Such a procedure might have obviated the only fatality in our series (Case 10) and would probably have shortened the course of illness in several of the cases (4, 7, and 8).

TULAREMIA

Our treatment of acute tularemia with streptomycin has corroborated previous reports of the effectiveness of this therapeutic agent.^{1, 29, 30, 31, 33} The antibiotic has shortened the duration of the disease and hastened resolution of the lymphadenitis. Two patients with tularemia have been treated with streptomycin. Both were of the ulceroglandular type, and both recovered rapidly after streptomycin treatment was begun.

J. C., a 10 year old white male, was hospitalized for twenty-two days. He entered with the history of being bitten on the finger by a cat four weeks previously. Twelve days later he developed a fever of 103° F. and was treated with sulfathiazole for three days. The temperature dropped to normal to recur three days later when the finger again became swollen, and lymphadenitis of the epitrochlear and axillary nodes developed. On physical examination there was a large oval ulcer on the fifth finger of the left hand. There were a large fluctuant mass over the medial lower portion of the left humerus and discrete enlarged submaxillary, supraclavicular, right epitrochlear and inguinal nodes. Laboratory work revealed a white blood cell count of 8850; Wassermann, darkfield and tuberculin tests were negative. Agglutination to *B. tularensis* was positive in 1:640. Therapy consisted of streptomycin 1.6 gm. per day for seventeen days. The boy was afebrile after three hospital days. The epitrochlear lymphadenitis became fluctuant on the ninth hospital day. The abscess was aspirated and 100,000 units of streptomycin were instilled. This was repeated on the fourteenth hospital day. On both occasions cultures were negative. The ulcer on the finger healed rapidly.

D. S. was an 11 year 8 months old white male. One week after shooting and dressing a rabbit he became ill with fever and chills and severe headaches occurring about four times a day. Several days later an ulcer appeared on his left hand, and the left axillary nodes became enlarged. At this time his mother became ill and was hospitalized with a diagnosis of tularemia. On physical examination the boy appeared acutely ill, and there was general glandular enlargement with an especially large node in the left axilla. A small ulcer was noted on the left hand. Laboratory data revealed a white blood cell count of 8700 and a 1:20 agglutination to *Pasteurella tularensis*. On the ninth hospital day the agglutination to *P. tularensis* was 1:1280. The blood culture was negative. The hospital course was uneventful. He was given 1.9 gm. of streptomycin a day for seven days with resolution of the axillary lymphadenitis. There were no febrile periods.

NONTUBERCULOUS PULMONARY INFECTION

Streptomycin was at first utilized in cases of pulmonary disease caused by organisms proved to be sensitive to the antibiotic.^{10, 35} On occasions when either toxic responses occurred with sulfonamides, or adequate doses of these compounds had been given without results, or when recurrence of pneumonic processes took place, streptomycin was utilized. In other instances there was hesitancy to start sulfonamides

because of the severe degree of dehydration and anhydremia in association with the pneumonia. This occurred chiefly in the younger age groups, and in order to furnish broad coverage the antibiotics were utilized until the causative organism could be identified and sensitivity studies made.

In this series, twenty-three cases of pulmonary disease other than tuberculosis were treated with streptomycin. Of these, fourteen cases were *bronchopneumonia* either acute or recurrent, and nine were cases of *acute bronchiolitis*. Several cases of *pancreatic fibrosis with chronic pneumonitis* were treated with questionable results. The cultures obtained showed the usual pathogens—*pneumococcus*, *staphylococcus* and *streptococcus*. Typical case summaries are presented below.

P. A. C., an 18 months old white female, had a cough and intermittent fever for one week prior to hospitalization for increasing respiratory difficulty. On physical examination there were purulent conjunctivitis, bilateral otitis media, and signs consistent with a pneumonic process in the right lower chest. Fluoroscopy substantiated the findings of pneumonia at the right base. The blood culture was negative. On therapy with penicillin and sulfadiazine, the temperature of 39.8° C. subsided by lysis to normal in twenty-four hours, and the general physical state improved. On the third hospital day the fever returned; sulfadiazine was discontinued, and streptomycin was given. Temperature was normal in twenty-four hours. Four-tenths gram of streptomycin was given over a three day period. Recovery was unevenful.

H. C., a 2½ year old white male, was admitted with a history of cough, hoarseness and fever for two weeks. Four days previous to admission he received adequate doses of sulfadiazine with no clinical improvement. He was moderately ill, and numerous rhonchi were heard throughout both lung fields. On fluoroscopy there was increase in hilar markings with suggestion of early central pneumonia in the right upper lobe. Immediate therapy consisted of penicillin and sulfamerazine, but a rash appeared in four hours, and the sulfonamides were discontinued and streptomycin substituted. Recovery was without incident.

C. L. P., a 7 months old white female, was hospitalized with a history of wheezing for three months, cough for four weeks and fever of one week's duration. She was placed on adequate amounts of sulfadiazine for one week before hospitalization with no relief. There was a known exposure to tuberculosis. Physical examination revealed a chronically ill infant in moderate respiratory distress. There were coarse rales throughout both lung fields. Therapy consisted of penicillin and sulfadiazine, and for four days there was no response. At this time fluid was noted in the left chest, and a thoracentesis was done. Cultures of this fluid for all bacteria were negative. On the fifth hospital day streptomycin was started, and the temperature dropped to normal within forty-eight hours. She received 2.1 gm. of streptomycin over a six day period, and following discontinuance there was no relapse. Tuberculin tests were negative as were animal inoculations with pleural fluid.

L. M. W., 17 months old white female, was admitted with recurrence of hacking paroxysmal cough associated with dyspnea, fever and vomiting of forty-eight hours' duration. This was the third attack. She had been hospitalized two weeks previously for the same symptoms at which time there was an unevenful recovery with use of penicillin. On physical examination the child was cyanotic. Respiratory excursions were rapid and shallow with prolonged expiration and rhonchi throughout both lung fields. The percussion note was hyperresonant. Fluoroscopy revealed

brilliant lung fields with low diaphragms and diminished excursions. On treatment with penicillin and general symptomatic measures the infant became cyanotic, and temperature elevation persisted. Fluoroscopy revealed an area of increased density at the left base. Streptomycin was then given. The temperature became normal in twenty-four hours, and marked clinical improvement occurred. The child made an uneventful recovery without recurrence of symptoms.

TUBERCULOUS INFECTIONS

In controlled experiments Feldman and Hinshaw^{40, 41} and many others have demonstrated that streptomycin protects guinea pigs against tuberculosis by exerting an inhibiting or suppressive effect on the growth of *Mycobacterium tuberculosis*. There are now numerous reports in the literature on the use of streptomycin in human tuberculous infections.^{19, 27, 42, 50, 60} These suggest that only certain clinical types of tuberculosis will respond favorably to the drug. The presence or development of streptomycin-resistant strains⁶⁴ of *Myco. tuberculosis* is frequently responsible for failure of streptomycin therapy. The consensus of most investigators is that tuberculosis of the non-fibrotic type responds most favorably to streptomycin therapy,^{41, 43, 63} that tuberculous meningitis and miliary tuberculosis when treated early and vigorously with streptomycin may be brought to an apparent state of arrest,^{11, 14, 18, 28, 40, 48} that resistant strains of *Myco. tuberculosis* usually develop by the end of a ninety day period, and that after the development of drug resistance specific therapy does not alter the progression of the disease. The major value of the drug lies in its temporary suppression of infection which permits healing of the disease by natural repair mechanisms.⁴³

Seven patients with tuberculous infections have been treated with streptomycin at this hospital.

D. K., a 2 months old colored female, was in intimate contact for twenty-three days with her tuberculous mother. She developed purulent discharge from the left ear and marked bilateral enlargement of the cervical lymph nodes. Roentgenograms of the chest showed a widening of the mediastinum. *Mycobacterium tuberculosis* was cultured on Petroff's medium from the aural exudate. The infant was given a total of 23.1 gm. of streptomycin with no improvement and died on the fifty-fourth day of treatment. Blood levels of streptomycin varied from 50 units one-half hour after administration of a 50,000 unit dose to less than 10 units three and one-half hours after a similar dose. At the time of maximum streptomycin blood level, the spinal fluid level was also found to be 33 to 50 units per cc., although no streptomycin had been administered by the intrathecal route.

D. McC., a 7½ months old white male, was first admitted to the hospital because of "failure to do well" and cough since the age of one month. His father suffered from active pulmonary tuberculosis. Physical examination revealed a poorly developed and nourished infant with a draining ear. Tuberculin test, 1:1000, was positive. An x-ray diagnosis of pulmonary tuberculosis, childhood type, involving all lung lobes, was made. General symptomatic measures were instituted. The child's general state improved, but aural discharge persisted. He was discharged home.

He was readmitted two months later with the same complaints. At this time

streptomycin ear drops (1 cc = 0.025 gm streptomycin) were utilized with slight diminution in the amount of aural discharge. He was again sent home.

Two months later the child returned to the hospital because of stiff neck and periods of cyanosis. The physical examination showed an acutely ill infant with nuchal rigidity and bilateral aural discharge. Liver and spleen were both palpable. Lumbar puncture revealed cerebrospinal fluid with 205 cells, 80 per cent lymphocytes, positive Pandy, and sugar of 15 mg per 100 cc. No organisms were seen. A smear of the aural discharge was positive for acid fast organisms. A punch biopsy of the liver showed typical tubercles. Streptomycin was administered intrathecally and subcutaneously for six days with no appreciable change in the cerebrospinal findings. The infant expired on the sixth day of treatment. Autopsy revealed caseous tuberculosis, right upper lobe, right middle lobe, tracheobronchial and mediastinal lymph nodes, miliary tuberculosis of the lungs, liver, spleen and kidney, and tuberculous endocarditis of the mitral valve.

C. J., a 3 weeks old colored female, hospitalized because of vomiting and increasing irritability showed on physical examination evidence of weight loss, Biot breathing and nuchal rigidity. Cerebrospinal fluid contained 890 cells per cubic mm., 93 per cent lymphocytes and acid fast organisms on smear. The spinal fluid sugar was 27 mg per 100 cc. when the blood sugar was 125 mg per 100 cc. One hundred thousand units of streptomycin intrathecally daily were started with 1 gm of streptomycin daily in divided doses subcutaneously. There was no marked improvement, and facial paralysis developed on the eleventh hospital day. On this day temperature was normal, but muscle twitches were noted. There was difficulty in obtaining spinal fluid, and a subarachnoid block developed during the second week. Because of this, the streptomycin was instilled in the ventricles. No untoward effects were noted until the thirty ninth hospital day when an intraventricular instillation was followed by high fever and convulsions. A repeat ventricular tap showed grossly bloody fluid. The infant had repeated convulsive seizures and died three days later. Autopsy revealed calcified caseous nodules in the lung and tracheobronchial lymph nodes and basilar tuberculous meningitis. There was clotted blood in both ventricles.

Therapy was probably adequate. Death appeared to be due to intraventricular hemorrhage. The meningitis had improved as evidenced by a fall in cerebrospinal fluid cell count to 42 and a rise in the cerebrospinal fluid sugar to 69 mg per 100 cc.

M. O., a 9 months old white female, began to vomit twelve days before admission and for six days did not "respond well." Physical examination revealed an obtunded infant. The liver was enlarged. Tuberculin test was positive; the cerebrospinal fluid contained 240 cells/cu. mm., and the glucose level was 10 mg per 100 cc. Streptomycin was administered intrathecally and subcutaneously for thirteen days with no apparent benefit. The infant expired on the thirteenth hospital day.

B. J. D., a 2½ year old white female, was hospitalized because of persistent severe cough, fever and swelling of the arms, legs and abdomen. She appeared acutely ill, breathing rapidly and shallowly. There was puffiness of the face, legs and arms. The lung fields were resonant, but numerous rales were heard throughout. By the fifth day the child was much worse. The abdomen was distended with fluid which on paracentesis revealed acid fast organisms. Two hundred thousand units of streptomycin were instilled, and the child was treated with streptomycin administered subcutaneously. On the seventh day she developed cardiac failure and required digitalization. There was gradual improvement noted by the end of the third week. On treatment with 1 gm of streptomycin a day, she developed questionable and transitory labyrinthine symptoms. During the next three weeks she improved rapidly. X-rays of the chest confirmed the diagnosis of pulmonary tuberculosis as did cultures of the sputum.

from the urine following a febrile reaction and were shown to be insensitive to streptomycin but inhibited by sulfadiazine. Although the patient remained afebrile pyocyanus was cultured from the urine repeatedly and was present until the urinary obstruction was relieved surgically and the elongated ureters resected.

Seven patients with *acute infections of the urinary tract* have been treated. Four were due to single bacterial infections, three of which were *E. coli* and one *Aerobacter aerogenes*. Clinical improvement was rapid with decrease in fever, leukocytosis and pyuria with subsequent sterilization of the urine. There was no bacteremia in this group. One patient received sulfadiazine but developed hematuria, streptomycin was substituted and the good response to sulfadiazine maintained.

There were three patients with *combined infection*

M S a 10 day old female while being treated with penicillin and streptomycin for *Staphylococcus albus* bacteremia developed a urinary tract infection due to *Staph. albus* and a streptomycin resistant strain of *B. coli*. The *B. coli* was sensitive to 50 u/cc. of streptomycin while *Staph. albus* was inhibited by 0.5 u/cc. There was an associated diminution in renal function and as soon as this improved pyuria cleared and cultures became negative. A total of 4.5 gm. of streptomycin was given over ten days.

M B 6 weeks old white male developed pyelonephritis due to *E. coli* *Staph. albus* and a nonhemolytic streptococcus. On a regime of penicillin and streptomycin there was rapid clearing of the urine, and cultures were sterile on the fifth day of therapy. He received 2.65 gm. of streptomycin over an eight day period.

D H. was a 9 months old white female whose urine revealed infection with *E. coli* and *Staph. albus*. Treatment with penicillin alone failed to eliminate *E. coli* and on streptomycin the staphylococcus was uncontrolled. Combined therapy with streptomycin and penicillin sterilized the urine after eight days.

It is, therefore, apparent that in all cases of urinary infection it is necessary to determine the nature of the infection and the sensitivity of the organism and to determine if underlying anatomic abnormalities of the genitourinary tract exist. It is obvious that all infecting organisms must be identified, their sensitivities determined and complete chemotherapeutic and antibiotic coverage provided if early cure is to be obtained (Cases M S, M B and D H). The presence of multiple renal calculi or of obstructing congenital anomalies contributes to failure of treatment and often permits the infecting organism to acquire streptomycin resistance (Cases P A R, J F, R B and W F). In two instances (Cases H C and J F) in which genitourinary anomalies exist and remain uncorrected and in another (R. B) where inoperable renal calculi are present, recurrent attacks of urinary infection have been encountered. In two cases in which the underlying anomaly has been corrected and obstruction relieved (Cases P A R. and W F), relapse has not occurred.

centration of streptomycin. Streptomycin was given, and surgical intervention to relieve ureteral obstruction due to adhesions and aberrant vessels was carried out. The infection cleared after elimination of the obstructing band and has not recurred.

H. C., a 7 months old white male, was admitted to the hospital because of diarrhea, dehydration and acidosis. Because of his general appearance, it was felt that some underlying disturbance was responsible for his illness. Further history revealed that the infant dribbled urine continuously. A diverticulum of the urethra was demonstrated on urethrography. During his acute illness, streptomycin and penicillin were administered with relief of symptoms. Four months later he was readmitted to the hospital with diarrhea, fever and constant urinary dribbling. *Bacillus proteus* was isolated from the urine and found to be sensitive to 0.5 u./cc. of streptomycin. He received 2.7 gm. of streptomycin over a seven day period with gradual improvement and sterilization of the urine. The congenital anomaly is still uncorrected.

J. P., a 2½ year old white male, was admitted to the hospital because of recurrent attacks of pyelonephritis and congenital absence of the left kidney. The first attack of pyelonephritis responded to penicillin and sulfadiazine. There was, however, a recurrence of symptoms, and streptomycin was started on the third hospital day when urine cultures revealed *Proteus vulgaris* sensitive to 0.5 u./cc. of streptomycin. He was given 3.375 gm. of streptomycin together with sulfapyrazine over a seven day period. There was marked clearing of the urine with no recurrence after six months.

J. F. was first admitted to the hospital with typical history and urinary findings of pyelonephritis. Cultures at this time revealed staphylococcus and *E. coli*. Pyelography showed double ureter and double pelvis on the left. The child received penicillin, sulfamylon and streptomycin for eight days with gradual clearing of the urine. On the seventeenth hospital day streptomycin was again given because of recurrence of pyuria, and she received 5.3 gm. over a seven day period. There was improvement, but four months later she suffered a recurrence. Urine culture revealed a strain of aerobacter which grew in 50 u./cc. of penicillin and 50 u./cc. of streptomycin. Even on very large doses of penicillin and streptomycin there was only gradual and incomplete clearing of the urine.

R. B., a 12 year old white female with inoperable bilateral renal calculi, was hospitalized on five occasions for recurrent attacks of pyelonephritis. In one instance the infection was due to a strain of *E. coli* sensitive to 0.5 u./cc. of streptomycin, on one occasion to infection with pyocyaneus. Both of these attacks subsided promptly after treatment with streptomycin was instituted. Three attacks were due to mixed infection. The first was due to *B. alcaligenes* and *E. coli* both sensitive to 0.5 u./cc. of streptomycin, and prompt recovery followed streptomycin therapy. In an attack in which *Ps. aeruginosa* sensitive to 50 u./cc. of streptomycin and pyocyaneus insensitive to 56 u./cc. were recovered, the urinary infection failed to respond to treatment with streptomycin and sulfamylon. This combined treatment also failed to cure an attack due to mixed infection with *E. coli* and pyocyaneus.

W. F., a 22 months old white male, was first hospitalized with a history of polyuria, polydipsia and enuresis for most of his life. Two days prior to admission there were fever, vomiting and dysuria. Examination revealed urethral obstruction at the bladder neck, enlarged bladder, and markedly dilated and tortuous ureters. Pyuria due to *E. coli* was demonstrated and was not eliminated by treatment with penicillin and sulfadiazine. Twelve days later the temperature rose to 41° C., and urinary culture revealed *E. coli* sensitive to 0.1 u./cc. of streptomycin and a strain of pyocyaneus sensitive to 0.5 u./cc. After two days of streptomycin treatment, the temperature was normal. One month later *B. coli* and pyocyaneus were isolated

from the urine following a febrile reaction and were shown to be insensitive to streptomycin but inhibited by sulfadiazine. Although the patient remained afebrile, pyocyanus was cultured from the urine repeatedly and was present until the urinary obstruction was relieved surgically and the elongated ureters resected.

Seven patients with *acute infections of the urinary tract* have been treated. Four were due to single bacterial infections, three of which were *E. coli* and one *Aerobacter aerogenes*. Clinical improvement was rapid with decrease in fever, leukocytosis and pyuria with subsequent sterilization of the urine. There was no bacteremia in this group. One patient received sulfadiazine but developed hematuria; streptomycin was substituted and the good response to sulfadiazine maintained.

There were three patients with *combined infection*.

M. S., a 10 day old female, while being treated with penicillin and streptomycin for *Staphylococcus albus* bacteremia developed a urinary tract infection due to *Staph. albus* and a streptomycin-resistant strain of *B. coli*. The *B. coli* was sensitive to 50 u./cc. of streptomycin while *Staph. albus* was inhibited by 0.5 u./cc. There was an associated diminution in renal function, and as soon as this improved pyuria cleared and cultures became negative. A total of 4.5 gm. of streptomycin was given over ten days.

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It is, therefore, apparent that in all cases of urinary infection it is necessary to determine the nature of the infection and the sensitivity of the organism and to determine if underlying anatomic abnormalities of the genitourinary tract exist. It is obvious that all infecting organisms must be identified, their sensitivities determined and complete chemotherapeutic and antibiotic coverage provided if early cure is to be obtained (Cases M. S., M. B. and D. H.). The presence of multiple renal calculi or of obstructing congenital anomalies contributes to failure of treatment and often permits the infecting organism to acquire streptomycin resistance (Cases P. A. R., J. F., R. B. and W. F.). In two instances (Cases H. C. and J. F.) in which genitourinary anomalies exist and remain uncorrected and in another (R. B.) where inoperable renal calculi are present, recurrent attacks of urinary infection have been encountered. In two cases in which the underlying anomaly has been corrected and obstruction relieved (Cases P. A. R. and W. F.), relapse has not occurred.

MISCELLANEOUS INFECTIONS

A heterogeneous group of cases was treated with streptomycin with variable results. Two cases of *osteomyelitis* in young infants—one five days and the other one month of age—were treated. In the younger a destructive process at the distal ends of both femora was found on x-ray. Penicillin and sulfadiazine were both given for three days with no change in the infant's course. Due to the persistence of fever, streptomycin was administered, and within twenty-four hours the temperature was normal, and gradual healing of the osteolytic process in the femora began. A total of 13.95 gm. of streptomycin was given over a seventeen day period. No evidence of toxicity was found. The other infant had an osteomyelitis of the right femur from which *E. coli* and a gamma streptococcus were cultured. The *E. coli* was sensitive to 10 u./cc. of streptomycin, and the gamma streptococcus failed to grow in media containing 0.1 u./cc. of penicillin. The course was afebrile after twenty-four hours. A total of 3.9 gm. of streptomycin was employed over a fifteen day period, and penicillin was given concomitantly.

Streptomycin was administered in one case of *lupus erythematosus*, two cases of *generalized histoplasmosis*,⁵⁸ and two cases of *encephalitis* of undetermined etiology with no effect clinically on the diseases. It was felt in the latter cases that the drug was of value in combination with penicillin in preventing complications arising as a result of the prolonged immobilization.

Further treatment of cases of *Caffey's syndrome* (infantile cortical hyperostoses) with streptomycin would be necessary to evaluate the response in one infant with typical clinical and roentgenological findings of this disease. After an ineffective course of penicillin and sulfadiazine, streptomycin (1.8 gm.) was given over an eight day period. The temperature dropped to normal, and there was a regression of the mandibular swelling. This may have been coincidental.

A 12 year old white female child presented a difficult dermatological problem. There was a five week history of *recurring crops of vesicles* coalescing to form large blebs intermingled with an acute diffuse erythematomacular eruption. Prior to hospitalization penicillin, sulfonamides, lotions, ointments and local irradiation were employed with no benefit. Following hospitalization penicillin was again unsuccessfully used. Cultures from blood and vesicles were negative. On utilizing streptomycin subcutaneously 1 gm. per day in divided doses, the skin cleared dramatically in the course of seven days. There has been no recurrence.

Bradford has utilized streptomycin in clinical cases of *pertussis*. Two infants of 6 and 11 months had pertussis for two and three weeks respectively. Under combined therapy with streptomycin and penicillin there was general improvement in these infants, and no complications

developed. Further investigation with streptomycin alone is necessary before clinical evaluation is possible.²⁷

Because of the sensitivity to streptomycin of many of the organisms constituting the normal bacterial flora of the bowel and the known efficacy of the drug in some cases of *peritonitis*,¹⁹⁻²⁰ it has been employed in treatment of peritonitis and in cases where infection of ascitic fluid by intestinal organisms has been suspected. In three cases of peritoneal infection following intestinal obstruction (congenital bands, intussusception), unexpectedly rapid convalescence has followed its use, and in two cases where peritoneal contamination was thought to be present or imminent, the drug was similarly effective. Infection of the ascitic fluid with *B. coli* in cases of chronic glomerulonephritis is believed to be a relatively common occurrence. It has been our practice to treat all such infections with streptomycin and penicillin until the infecting organism is identified.

CONCLUSIONS

Results at the St. Louis Children's Hospital of the use of streptomycin in influenzal meningitis, tularemia, tuberculosis and urinary infections have paralleled those previously reported from other clinics.

The use of streptomycin administered orally and parenterally in cases of infantile diarrhea appears to decrease further the mortality rate in this disease and to speed recovery.

In severe infections of infancy and childhood (e.g., meningitis, septicemia) it appears desirable to provide the broadest antibacterial coverage possible until the infecting agent is isolated and its sensitivities determined.

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Patients with chronic glaucoma and with symptoms of headache, supraorbital pain, sense of fullness of the eyes or brows, often accompanied by visual disturbances, not uncommonly give the ophthalmologist a history of treatment for other diseases which characteristically exhibit similar complaints. Most frequent among these are (1) chronic sinusitis, (2) refractive errors and eyestrain, (3) menopause or menstrual headaches, (4) conjunctivitis, (5) neurasthenia, (6) migraine, (7) neuralgia, (8) vascular hypertension and (9) chronic nephritis. Less frequent are early uremia, brain tumor, iritis, syphilitic meningitis, and multiple sclerosis. A moment's re-

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When chronic glaucoma is detected early, treatment is in many cases very effective and prognosis is good. Miotic drops such as pilocarpine, eserine, prostigmine, acetylcholine, carbachol alone or in combination, often serve to reduce intraocular tension and, in many cases, prevent further deterioration of the visual fields. While the ophthalmologist must regulate the strength and frequency of these drugs by periodic check on the tension and fields, the family doctor, with his superior knowledge of the patient as an individual, of his home life and his emotional constitution, plays a very important part in the therapy of this disease. Here the psychosomatic factor is increasingly coming to be recognized, and most ophthalmologists have seen chronic glaucoma cases which should have done well, deteriorate because of adverse psychological circumstances.

If miotics do not control the glaucoma, surgery is indicated. Again, in the early cases the prognosis is usually good, but becomes poorer as the disease advances, particularly where there is any considerable degree of optic atrophy and field defect. It should be noted that, even in the most successful cases, little more can be achieved than the arrest of field loss as it existed at the time of operation. For example, a patient who has lost 50 per cent of his visual field does not regain this lost portion if successfully operated upon, but may retain that percentage which was present at operation. Where the disks are very pale, it is not unusual for the atrophy to continue to progress despite surgical control of ocular tension.

Most operations for glaucoma are designed to create an artificial channel for drainage of aqueous fluid out of the globe, thus acting as a sort of safety valve to maintain low intraocular pressure. The favorite method is to make an opening in the sclerotic coat, usually at the junction of sclera and cornea, underneath a flap of bulbar conjunctiva. Different techniques employ a minute trephine, a keratome or a small cataract knife and scissors; an iridectomy is often performed at the same time, and many surgeons leave a portion of iris in the opening to facilitate drainage and prevent the closure of the safety valve.

The operation is usually done under local anesthesia, inflicts little or no discomfort and may easily be performed at any age since the patient sits up in bed or even out of bed the following day. Very

among ophthalmologists that the disease is more commonly found among persons who are emotionally high strung and/or show evidence of vasomotor instability or vascular degenerative changes. The incidence of onset subsequent to excitement, anxiety, shock or grief is well known, and the time relation to the climacteric, both male and female, is present in a large percentage of cases.

All of the foregoing has the utmost diagnostic significance far beyond mere statistics, because it is in these very patients (the nervous, the anxious, the arteriosclerotic, the menopausal) that the physician will be confronted with complaints of headache and even symptoms directly referable to the eyes such as strain, burning, blurring, fatigue, supraorbital or retrobulbar pain—yet these need not necessarily be glaucomatous. To make judgment more difficult, the age of greatest incidence of glaucoma corresponds with the age of most marked presbyopic change, i.e., the progressive deterioration of near vision in the normal, healthy eye.

How, then, can we distinguish the glaucomatous patient from the purely nervous, menopausal, hypochondriac or presbyopic? Sometimes this is difficult in borderline cases, so that even the ophthalmologist is obliged to resort to provocative tests.

SYMPTOMS

Headache.—This is one of the most frequent symptoms of early chronic glaucoma and occasionally the only symptom which the patient will mention spontaneously. However, questioning may reveal certain characteristics which should arouse our suspicions. While usually supraorbital or frontal, on one or both sides, the headaches of glaucoma have a tendency to come on in the evening, particularly in the dark or in dim illumination as in motion picture theaters. Occasionally they will occur in the morning upon arising, usually in early cases, lasting less than one-half hour. They may be induced by vasomotor stimulus such as hot or cold bath, strong coffee, excitement. In most early cases the pain is not severe, and often is described only as a feeling of fullness or heaviness.

Vision.—Occasionally the patient reports no visual disturbance. On questioning, he will often recall haziness or fogginess of vision in one or both eyes, particularly under conditions mentioned above as tending to produce headache. In many cases (*and this is important*) they will definitely correlate headache and fogging. *Fairly consistent coexistence of these two symptoms may be considered presumptive evidence of glaucoma.* In this connection it should be noted that fogging of vision should be distinguished from blurring, since the latter has much less significance. To do this the patient should be asked whether his visual phenomenon resembles the sensation of a room filled with light mist or smoke (fogging) or that of a photograph out of focus (blurring).

An even more significant symptom is the *halo*. This is seen only at night when the patient looks at a small light such as a distant street lamp. Any normal eye with the lid half shut may be made to see rays emanating from the light and going *radially*. This is not a halo. A halo is a ring surrounding the light which usually consists of an outer red and an inner blue component. This will be seen only when the tension is sufficiently elevated to cause faint corneal edema, and is often accompanied by pain or sense of fullness in eye or brow. An excellent time to observe halos is on returning home in dark streets after seeing a motion picture. However, many glaucoma patients do not report halos.

Visual Acuity—It is of the utmost importance that the physician realize that many cases of chronic glaucoma do not show diminished acuity of vision on Snellen chart testing until very late in the disease. In fact it is not unusual for patients with advanced glaucoma and fields so shrunk as to render them almost helpless, still to read 20/20 or 20/30 on the office chart.

Visual Fields—Diagnostically, contraction of the visual field is second in value only to the actual finding of elevated intraocular tension by means of the tonometer. The early field defects may be shown by the more delicate methods used by the ophthalmologist, but unfortunately, when gross field changes are already demonstrable, the glaucoma is no longer in its early stages. However, assuming that a tardy diagnosis is better than none, the general physician should make it a practice to perform a *qualitative* test on the visual field of patients whose history is suspicious. A very approximate test, but one which may be carried out very quickly and with no special equipment is as follows.

The physician and patient stand confronting each other and about 8 feet apart, the patient having his back to the window or other source of light. If the test is performed in the evening, any lights overhead or in front of the patient should be extinguished leaving only whatever light comes from behind him. The patient is instructed to hold his left eyelid down with the forefinger of his left hand (keeping the other four fingers flexed so as not to obstruct the vision) and to look steadily with his right eye at the left eye of the physician facing him. The physician keeps his right eye closed and, with his left eye, fixes the gaze of the patient's open eye (the patient's right eye and the doctor's left eye are, of course, opposite each other as doctor and patient confront each other).

The patient is shown the test object which need be no more elaborate than a small florist's pin with a white head, preferably about 3 mm ($\frac{1}{8}$ inch) in diameter, attached to a thin black or dark gray rod about 12 inches long. He is instructed to say, "Yes" or "I see it," each time this white object is perceived by him at the edge of his visual field.

The physician then holds the object far to the side, as nearly as

possible on a plane midway between himself and the patient, and brings it slowly toward their common line of vision. If the patient's field is normal, he should perceive the object at the same point as the examiner. If the field is contracted in that meridian, he will perceive it somewhat further inward. This is repeated for all 12 meridians—12 o'clock, 1 o'clock, 2 o'clock, etc. Then the left eye is tested in the same manner—the left eye looking at the physician's right eye.

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The operation is usually done under local anesthesia, inflicts little or no discomfort and may easily be performed at any age since the patient sits up in bed or even out of bed the following day. Very

apprehensive patients may readily be subjected to operation under brief intravenous anesthesia.

SUMMARY AND CONCLUSIONS

1. Headache, attributed to any of the well known causes, occasionally turns out to be due to chronic glaucoma.

2. Headache may be the *only* symptom volunteered by a chronic glaucoma patient.

3. Careful questioning of such a patient may elicit other associated symptoms of visual disturbance, albeit transient and slight, such as fogging and halos.

4. A simple method is described whereby the physician may determine roughly and rapidly, and without special apparatus, whether there is any gross defect in the visual field.

5. Association of headache or ocular discomfort or foggy vision with gross field defect is presumptive evidence of chronic glaucoma.

6. Chronic glaucoma often insidiously goes on to blindness without any of the violent symptoms (inflammation, severe pain, vomiting) familiarly associated with *acute* glaucoma.

7. As with many other serious ailments, the difference between success and tragedy in chronic glaucoma depends upon *early* recognition and treatment.

8. In the interest of reducing the incidence of blindness, doctors are urged to include chronic glaucoma in their differential diagnosis of the causes of headache.

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